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BRONCHOSCOPIC OBSERVATIONS ON OBSTRUCTIVE PULMONARY ATELECTASIS *

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Keen interest was aroused throughout the medical profession in the pulmonary pathology and symptomatology produced by the sojourn of aspirated foreign bodies in the lung by the publications of Chevalier Jackson, Willis F. Manges and Thomas McCrae. Jackson's description of his bronchoscopic observation of the mechanics of the type of obstruction producing the "asthmatoid wheeze," obstructive emphysema and obstructive atelectasis, correlated with the roentgenologic observations of Manges and the physical signs of McCrae, gave a new conception of the part played in pulmonary disease by impairment of ventilation and drainage through the tracheobronchial tree. The result of the work has been a clinical and experimental revision of knowledge of many of the pulmonary diseases. This is especially true in pulmonary atelectasis. All physical signs, symptoms and roentgen observations of foreign body obstruction may be produced by pulmonary dysfunction and disease. The asthmatoid wheeze, obstructive emphysema and obstructive atelectasis may be the result of the same mechanism in disease as in foreign body. Partial obstruction producing the asthmatoid wheeze, expiratory obstruction producing emphysema and complete obstruction producing atelectasis is the sequence that usually results from the progressive increase in obstruction, the process finally resulting in drowned lung and pulmonary abscess.

Obstructive atelectasis may be termed massive when more than one lobe is involved, lobar when a single lobe is involved, and lobular when only the lobule of the lung is affected, the extent of atelectasis depending on the location of the obstruction in the tracheobronchial tree tributary to the area involved. Bronchial obstruction may be due to obstructive secretions, to an intra-bronchial lesion arising from the bronchial mucosa, to a lesion in the bronchial wall intruding into the bronchus, to disease of the peribronchial structures, to compression of the bronchus by disease processes from without or to a combination of two or more of these conditions. When the lesion is in the primary or secondary bronchi, it may be visible on direct bronchoscopic examination,

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and in many instances may be amenable to treatment by bronchoscopic intervention. We should, then, regard evidences of bronchial obstruction in diseases as a clear indication for bronchoscopic examination. Bronchoscopic observations in the following pulmonary conditions are submitted, with illustrative cases.

POSTOPERATIVE OBSTRUCTIVE ATELECTASIS (MASSIVE COLLAPSE OF THE LUNG)

The similarity of the roentgenologic and physical observations in postoperative massive collapse of the lung to the same observations in obstructive atelectasis due to foreign body was noted by Lee, Pancoast and Leopold. They compared their observations in massive collapse with the roentgenologic and physical observations of Manges, McCrae and Jackson in cases of foreign body. The privilege of first demonstrating by bronchoscopic examination the mechanism of postoperative massive collapse fell to my lot when, in January, 1925, I made such an examination of a patient of Dr. Walter Estell Lee. The cause of collapse was found to be complete obstruction of the main bronchus and its subdivisions tributary to the area of atelectatic lung by thick tenacious secretion.[1] The observations in this first case have been confirmed many times in the past five years by bronchoscopic examination in a large number of cases by various bronchoscopists. The lesion is most easily demonstrated by roentgenologic and physical signs in the right lung and apparently occurs more frequently on the right side. A case on the left side, which was definite and which cleared immediately after bronchoscopy, was reported by Dr. Calvin Smyth.[2]

A girl, aged 18, a nurse in training at the Abington Hospital, Philadelphia, had appendicitis. She was operated on under ether anesthesia, and thirty-six hours after the operation she presented the typical clinical picture of postoperative massive collapse of the lung. The roentgen examination showed the entire left lung to be involved (fig. 1 *A*). The heart was displaced entirely into the left side of the chest. The patient showed marked dyspnea and complained of a pulling pain in the region of the mediastinum. I was asked to examine this patient, and I aspirated bronchoscopically a considerable quantity of thick obstructive secretion from the left main bronchus and from the bronchi of the left upper and lower lobes and their subdivisions. The pain and dyspnea subsided immediately, and roentgen examination, twelve hours later, showed that the heart had gone back to practically its normal position (fig. 1 *B*). There was no recurrence of the collapse, and the patient's recovery was uneventful.

There are many factors, of course, in the mechanism of postoperative pulmonary collapse, but when the collapse has once occurred, the indication is to remove the obstructive secretion from the portion of the

1. Lee, W. E., and Tucker, Gabriel: Tr. Coll. Phys., Phil. **47**:231, 1925.
2. Smyth, Calvin: Ann. Surg. **90**:441 (Sept.) 1929.

tracheobronchial tree tributary to the area of atelectasis. This may be done by encouraging the patient to cough, by turning the patient on the opposite side and by "shaking" the patient as is sometimes advocated. All these measures should be tried. But in a certain percentage of the cases they are not sufficient, and bronchoscopy offers a positive means for removing the obstructing secretions. A most interesting piece of experimental work has been done by Van Allen,[3] showing the intercommunication between air vesicles in the same lobe of the lung. It helps to explain the mechanism of the clearing up of the lung in these cases following bronchoscopy. It is not possible to aspirate the obstructive secretion from every small bronchus, but when the air gets into a lobe of the lung and can find its way into the obstructed air vesicles, the patient can cough out the secretion that could not be removed at bron-

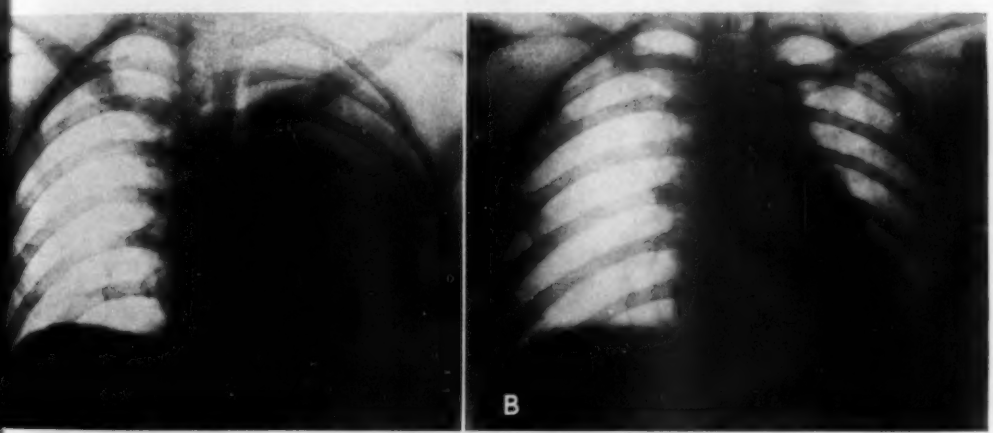

Fig. 1.—*A*, roentgenogram of the chest of a young woman, aged 18, showing atelectasis of the left lung and displacement of the heart into the left side of the chest; *B*, roentgenogram of the same patient, taken after bronchoscopic aspiration of the obstructing secretions from the left lung, showing the lung clear and the heart back nearly in its normal position. The young woman was a patient of Dr. Calvin Smythe.

choscopic aspiration. The essential thing, then, is that the larger bronchi be opened by aspirating the obstructing secretion, allowing the air to get in. As the cough mechanism is reestablished, the air is able to get into the periphery of the lung back of the secretion, and the secretion can then be coughed out. A complete bibliography on postoperative atelectasis has been compiled by Bowen.[4]

3. Van Allen, C. M., and Lindskog, G. E.: Obstructive Pulmonary Atelectasis: Problems of Pathogenesis and Clinical Management, Arch. Surg. **21**:1195 (Dec.) 1930.

4. Bowen: Am. J. Roentgenol. **21**:101, 1929.

BRONCHIAL ASTHMA, MASSIVE OBSTRUCTIVE ATELECTASIS (NOT POSTOPERATIVE)

All bronchoscopists have noted the similarity of secretions in certain cases of asthma and in cases of postoperative atelectasis. It has been noted by Wilmer, Cobe and Lee,[5] that there is a definite relation between allergy and postoperative atelectasis. A large percentage of Lee's patients with postoperative atelectasis showed definite allergy. Atelectasis has previously been observed in asthmatic persons. The case described in the following paragraph is, however, the first in which the actual mechanism of atelectasis in asthma was demonstrated bronchoscopically.

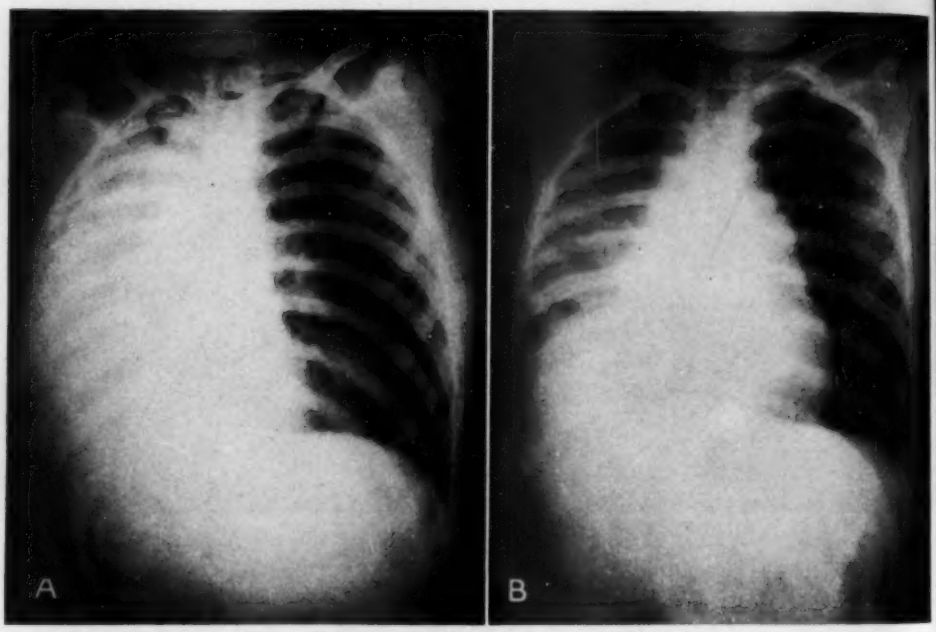

Fig. 2.—*A*, roentgenogram of the chest of a girl, aged 12, a patient of Dr. Harry Wilmer and Dr. Walter Estell Lee, taken twenty-four hours after the onset of an atelectasis that occurred during an attack of asthma (not postoperative), showing atelectasis of the right lung, the lower and middle lobe and a portion of the upper lobe, with a displacement of the heart to the right; *B*, roentgenogram of the same patient after bronchoscopic aspiration of obstructing secretions. The lung has largely cleared, and the heart has gone back to the left.

A girl, 12 years of age, a patient of Drs. Wilmer and Lee, during an asthmatic attack presented atelectasis of the right lung. The lesion which was demonstrated on physical and roentgen examination (fig. 2*A*) gave a typical picture of collapse on the right side, such as is seen in postoperative cases. At the request

5. Wilmer, H. B.; Cobe, H. M., and Lee, W. E.: Ann. Surg. **91**:651 (May) 1930.

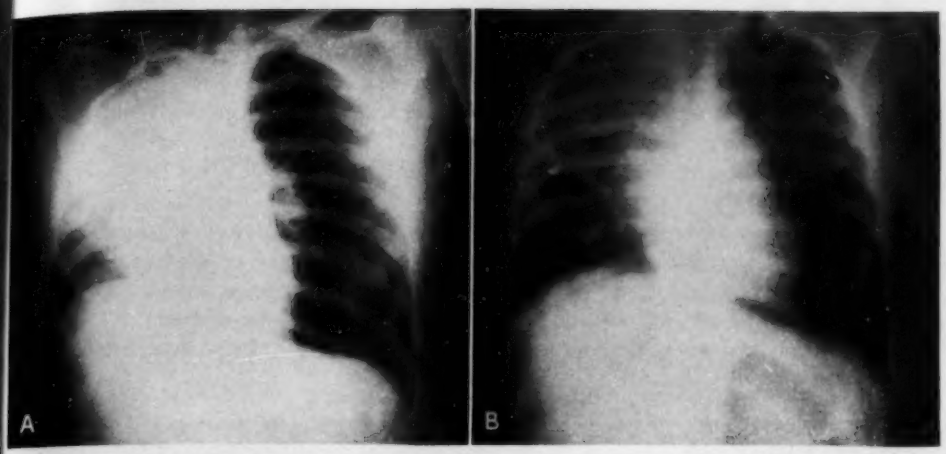

Fig. 3.—*A*, roentgenogram of the same patient as in figure 2, showing a recurrence of the atelectasis in the middle lobe and a portion of the upper lobe, with displacement of the heart to the right and marked elevation of the diaphragm on the right. This film was made forty-eight hours after the first bronchoscopic aspiration. *B*, roentgenogram of the same patient, after the second bronchoscopic aspiration; the lung has largely cleared, and the heart has gone back to almost its normal position.

Fig. 4.—Roentgenogram of the same patient as in figures 2 and 3 two days after the second bronchoscopy, showing the heart and the lung in normal position. There were no signs of atelectasis on physical examination. The roentgenograms were taken by Dr. Thomas Loughrey.

of Drs. Wilmer and Lee, I made a bronchoscopic examination of this patient and found the mechanism of the condition identical with that in postoperative massive collapse. A large quantity of obstructive secretion was aspirated, with a partial clearing of the lung immediately (fig. 2*B*). After forty-eight hours there was a gradual recurrence of the atelectasis limited to the upper and middle lobes (fig. 3*A*). A second bronchoscopy was done, and again a considerable quantity of obstructive secretion was removed from the bronchus of the middle lobe and of the upper lobe of the right lung. The main bronchus, however, was not completely obstructed. Following the second bronchoscopy, the lung cleared promptly (fig. 3*B*), and after forty-eight hours was entirely clear, the asthmatic attack having subsided (fig. 4).

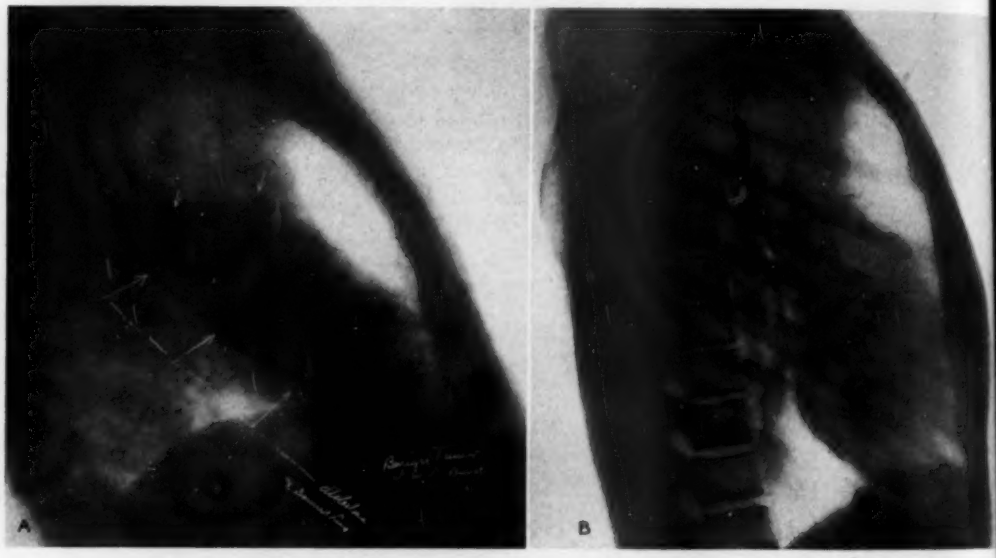

Fig. 5.—*A*, roentgenogram of a man, 57 years of age, showing an area of atelectasis and drowned lung on the lower portion of the upper lobe of the left lung. The roentgenogram was made before the bronchoscopic removal of an obstructing tumor from the bronchus. *B*, roentgenogram of the same patient one year after the removal of a tumor from the bronchus of the left upper lobe and after the suppurating lesion of the lung had cleared up. The roentgenograms were taken by G. E. Pfahler and J. Gershon Cohen.

BENIGN INFLAMMATORY NODULE OF THE BRONCHUS, PRODUCING OBSTRUCTIVE ATELECTASIS WITH DROWNED LUNG

A man, 57 years of age, was admitted to the Graduate Hospital, University of Pennsylvania Chevalier Jackson Bronchoscopic Clinic, with a diagnosis of bronchiectasis and pulmonary abscess. There was a history of an acute onset of illness which was diagnosed as influenza and which was followed by purulent expectoration, wheezing respiration and, on physical and roentgen examination three months later, an area of pathologic change in the left lung. On admission roentgen examination showed a triangular area of density in the anterior portion of the

lower left side of the chest (fig. 5*A*). On bronchoscopic examination, the stream of pus was followed down into the left main bronchus and seemed to be coming from the bronchus of the left upper lobe. A grayish, smooth, nodular mass was protruding from the bronchial orifice of the left upper lobe. It had the appearance of tumor, and a section was removed for histologic examination. A large quantity of pus was aspirated from the branch bronchi above the location of the tumor, which was blocking completely the lower branch of the bronchus of the left upper lobe. On histologic examination, the tissue removed appeared benign. Further bronchoscopic removal was done, and a mass the size of the finger was enucleated from the bronchus of the left upper lobe. It left a smooth surface with no bleed-

Fig. 6.—Photomicrograph of a section of the benign inflammatory nodule removed from the bronchus of the left upper lobe of the patient in figure 5; it shows calcareous deposits and true bone formation. The microscopic section was made by Dr. E. A. Case.

ing, and a large quantity of pus was aspirated from the branch beyond the obstructing tumor. The temperature immediately became normal; expectoration of pus continued for several days. The roentgen examination showed that the area of density in the lower portion of the left upper lobe had largely disappeared. The man's condition became normal (fig. 5*B*). Histologic examination of the tumor was reported by Dr. E. A. Case as follows:

"The surface of this mass is partly covered with epithelium and partly ulcerated. The epithelium is of the stratified columnar type, but in a very few small places it assumes squamous characteristics. In the center of the mass there is a piece of

bone, about 5 mm. by 3 mm. It is elliptical in outline, and the center contains a very cellular tissue with many polymorphonuclears, mononuclears, lymphocytes and cells resembling bone-marrow cells, including giant cells. About this bone the tissue is fibrous, ranging in density from very loose edematous material to areas suggesting cartilaginous development. Small masses of fatty tissue are present. Glands dip in from the surface epithelium. Where the surface epithelium is lost, there is a covering of fibrin with blood and leukocytes.

"Diagnosis: It is hard to place this growth. I am of the opinion that it is primarily an inflammatory process with bone formation."

CANCER OF THE LUNG, MASSIVE OBSTRUCTIVE ATELECTASIS

This patient was referred by Dr. Donald Ferguson, of Philadelphia, who previously reported the case.[6]

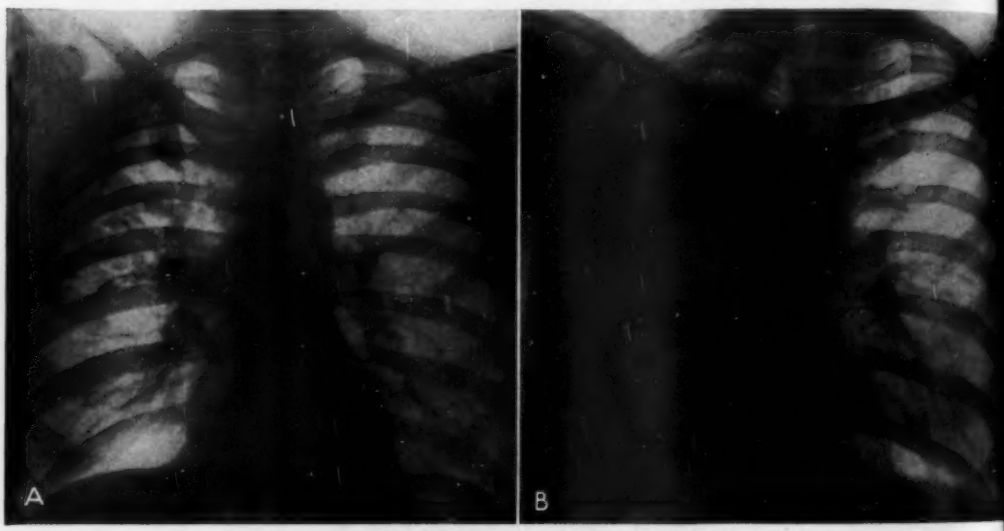

Fig. 7.—*A*, roentgenogram, showing peribronchial thickening and increase in hilus shadows in a man, 37 years of age, who complained of slight cough and wheeze, without hemoptysis; *B*, roentgenogram of the same patient, showing complete atelectasis of the right lung with displacement of the heart into the right side of the chest, with deflection of the trachea to the right. The bronchus was obstructed by a tumor mass, which was found on bronchoscopic examination to be cancer. The man was a patient of Dr. Donald Ferguson. The roentgenograms were taken by Dr. G. E. Pfhaler and Dr. J. Gershon Cohen.

A man, 37 years of age, of robust physique, a former football player, consulted Dr. Ferguson, complaining of a slight wheeze and an uncomfortable feeling in the chest. Roentgen examination (fig. 7 *A*) showed considerable peribronchial thickening with accentuation of the trunk shadows. One week later Dr. Ferguson was again consulted by the patient, who then complained of a severe tearing pain in the region of the heart and referred to the right side of the chest. He was

6. Ferguson, Donald: Hahneman. Monthly **64**:562 (Aug.) 1929.

urgently dyspneic, and roentgen examination showed complete atelectasis of the right lung with a displacement of the heart and mediastinum to the right side of the chest (fig. 7 *B*). Dr. Ferguson asked me to see this patient, and on bronchoscopy I found an infiltrating mass involving the right main bronchus and completely occluding its lumen. A specimen of tissue was removed bronchoscopically for histologic examination, and was reported by Dr. John A. Kolmer as cancer. Radiation therapy was used with palliative effect for three months.

BRONCHIAL STRICTURE, BRONCHIECTASIS, MASSIVE ATELECTASIS WITH FIBROSIS OF THE LUNG

A boy, 12 years of age, who had been a patient in a state sanatorium for tuberculosis was returned home with a report that in many examinations of the

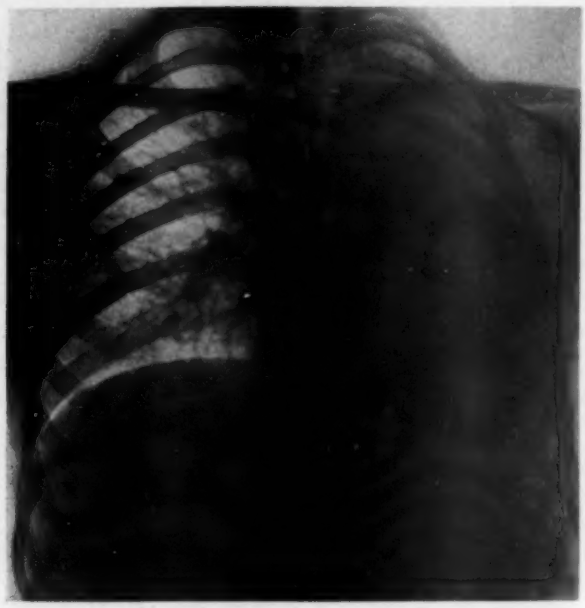

Fig. 8.—Roentgenogram of the chest of a boy, aged 12, showing atelectasis with fibrosis of the left lung and the heart displaced entirely into the left side of the chest.

sputum tubercle bacilli had not been found. The boy's general condition was good when he was returned home, and he entered school. Dr. J. Hart Toland, as medical consultant for the school, saw the boy. On physical examination, a massive atelectasis of the entire left lung was found. The picture was so typical of atelectasis that Dr. Toland thought of the possibility of foreign body and had him admitted to the Graduate Hospital for diagnostic study. At the suggestion of Dr. Toland and Dr. H. C. Carpenter, I was asked to see the boy in consultation, with a view to diagnostic bronchoscopy. Roentgen examination confirmed the observations of Dr. Toland and Dr. Carpenter. The heart was displaced entirely into the left side of the chest (fig. 8). On bronchoscopic examination, the left main bronchus was found completely occluded at a point about 1 cm. from the carina. The occlusion was funnel-shaped, and on introduction of a very small

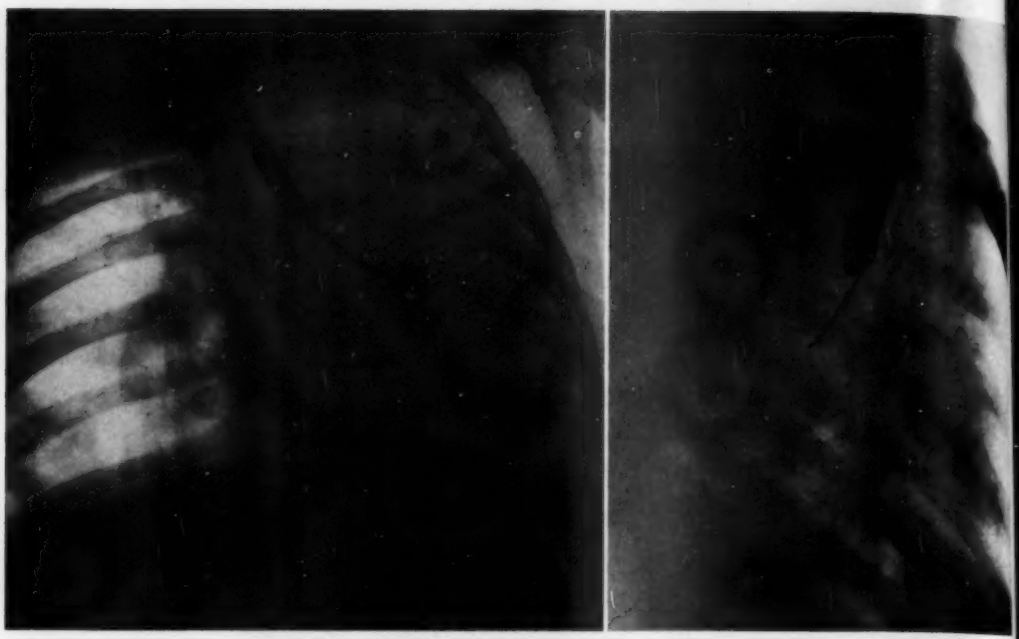

Fig. 9.—Roentgenograms of the chest of the same patient as in figure 8, showing the bronchoscope in the trachea with an opaque bougie introduced into the strictured bronchus of the left lung in both the anteroposterior and the lateral views.

Fig. 10.—Roentgenogram, showing the bronchoscope in position with a flexible-tipped injection tube in the left lung with the iodized oil that has been injected, revealing the bronchiectatic cavity in the left lung. The boy was a patient of Dr. Toland and Dr. Carpenter. The roentgenograms were taken by Dr. G. E. Pfhaler and Dr. J. Gershon Cohen.

flexible-tipped bougie, it was found to pass for a distance of about 3 cm. Considerable pus was aspirated; bacteriologic studies failed to show tubercle bacilli. The obstructed bronchus was gradually dilated (fig. 9) until a small aspirating tube could be introduced. Iodized poppy seed oil 40 per cent was then injected, and distinct bronchiectatic cavities were outlined (fig. 10). There was nothing in the history that would account for the massive lesion in the lung. A congenital stricture with atelectasis, infection, bronchiectasis and fibrosis of the lung would seem the most probable explanation of the condition.

CONCLUSIONS

Pulmonary disease may cause bronchial obstruction comparable in mechanism, physical signs and roentgen manifestations to the obstruction produced by aspirated foreign body.

When obstructive pulmonary atelectasis occurs, it means that complete occlusion of the bronchus tributary to the atelectatic area has occurred.

Obstructive pulmonary atelectasis is an indication for diagnostic bronchoscopy and in many cases an indication for bronchoscopic intervention for treatment, as well.

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ARTIFICIAL SUNLIGHT IN THE TREATMENT OF LARYNGEAL TUBERCULOSIS

MODIFIED CARBON ARCLIGHT *

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In the last few years, through the untiring energy of Dr. Wessely,[1] the university throat clinic—Hajekclinic—of Vienna has brought forth an ingenious method of treating tuberculosis of the upper air passages by means of concentrated artificial sunlight.

Before giving a detailed description of the cases of patients under my personal care from October, 1925, to October, 1928, I should like to record briefly what has been accomplished during the past sixty years in the treatment for laryngeal tuberculosis. Of course, local treatment of the larynx in any form or manner can be dated only from the discovery of the laryngeal mirror. The first attempts by pioneer laryngologists to treat the tuberculous larynx locally were summarized by Stoerk as a "noli me tangere" (touch me not). These words left a paralyzing influence for many years on further therapeutic attempts.

However, in 1876, M. Schmidt published a paper reporting the favorable influences obtained from the inhalation of antiseptic drugs, such as dilute solutions of phenol, balsam of peru, thymol, etc. This report was received with enthusiasm, especially when it was corroborated from many sources. Following these reports, other antiseptics, such as iodoform, iodol, creosote, menthol and antipyrine, some in concentrated solutions and others in powder form, were tried.

Complete statistics were compiled on the excellent results and improvements resulting from the use of each drug. The unfavorable results were only casually noted. To prove the value of menthol over

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* From the Department of Laryngology and Broncho-Oesophagoscopy, Service of Dr. Samuel J. Kopetsky, Beth Israel Hospital, New York.

1. Wessely, Emil: Eine neue Methode für die Behandlung der Tuberculose der oberen Luftwege mittels lokal applizierten kuenstlichem Lichtes, Wien. klin. Wchnschr. **37**:611, 1924; Die Behandlung der Tuberculose der oberen Luftwege mit kuentslichem Sonnenlichte, Monatschr. f. Ohrenh. **59**:1180, 1263, 1925. Weitere Erfahrungen über Behandlung der Schleimhauttuberculose mit künstlichem Sonnenlichte, Wien. med. Wchnschr. **77**:1115, 1927; Behandlung der Schleimhauttuberkulose mit künstlichem Sonnenlichte, Strahlentherapie **34**:798, 1929.

other antiseptics, one laryngologist went so far as to treat one side of the larynx with menthol and the other side with another antiseptic; he recorded the observation that the side treated with menthol regularly healed much sooner.

I need not mention the opinion held today concerning the use of antiseptic remedies in the treatment for laryngeal tuberculosis. Absolute proof is lacking that healing ever occurred under such treatment. Today applications of antiseptic remedies to bring about healing of tuberculous lesions in the larynx are little used.

The question then arises: How was it possible that many authors claimed to have observed lesions in the larynx healed by the use of the aforementioned antiseptics? To answer this question one must remember that prior to the publication of M. Schmidt's article, tuberculous lesions of the larynx were not treated. In advanced cases of laryngeal tuberculosis the lesions were covered with a filthy necrotic layer due to the passing of secretions from the infected lungs which caused a tremendous reaction in the surrounding tissues. There is little wonder, then, that following Schmidt's publication, the use of antiseptic sprays and powders to the affected larynx cleansed the lesions, the necrotic membranes disappeared, and the surrounding inflammatory swellings diminished to a certain extent. With these changes in the larynx, the patient's condition improved somewhat, his voice became temporarily clearer, and the agonizing dysphagia ceased for a while. Such objective and subjective improvements were readily interpreted as the initial stadium to recovery. While the observations were correct, the results were much exaggerated.

It is also true that a few superficial ulcers might have healed entirely, as there are many cases in which superficial ulcers healed without local medication, under a climatic, dietetic, hygienic regimen. But outside of their cleansing value in the great majority of cases the antiseptic measures did nothing to improve the lesions.

Of the great number of remedies that were tried locally in the treatment for laryngeal tuberculosis, lactic acid, introduced by Krause in 1885, for a long time enjoyed an almost specific reputation. It was hailed by laryngologists as the remedy par excellence, and although it is still being used by many of them today, no one even whispers about its curative properties.

In 1889 the so-called specific tuberculin was prepared, and a wave of enthusiasm broke out among the entire medical profession. But in the treatment for tuberculosis of the larynx this enthusiasm soon evaporated, and was thrown by the wayside. Small doses of tuberculin had no effect and large doses caused the lesions to advance rapidly and break down. This unsuccessful treatment was observed and metaphorically illustrated in those days by Schnitzler and Hajek.

Following endolaryngeal or extralaryngeal surgical procedures, the object of which is the sharp removal of tuberculous tissue from the diseased larynx, few if any encouraging results were reported. The "silent treatment," advocated by Sir St. Clair Thompson, calculated to put the larynx at rest, is based on sound physiologic reasoning. While giving good results in selected cases, this treatment is not adaptable to all patients; some must of necessity use their vocal apparatus in their various vocations, and others find it impossible to stop talking.

In order to conserve time and space I have merely mentioned the most important remedies and measures that have been used in the treatment for laryngeal tuberculosis. Drugs with or without analgesic properties and other devices intended to alleviate pain will not be considered here.

But before discussing the main subject of this paper, I shall mention the use of the galvanocautery. In 1907, Greenwald advocated the use of this instrument for piercing the infiltrated area and thus bringing about collapse and destruction of the lesion. While not the most popular form of treatment, it gives splendid results in a certain number of cases. But its use is limited to circumscribed lesions, such as superficial ulcers and circumscribed infiltrations. In the former, the flat burner touching the lesion is used; in the latter, the pointed one piercing the swelling. Until recently, the use of the cautery in laryngeal tuberculosis was considered the best form of treatment by all eminent laryngologists. Even to this day, in spite of the excellent results obtained with artificial sunlight, many famous laryngologists use that form of treatment to the exclusion of all others.

THE DEVELOPMENT OF THE USE OF CONCENTRATED ARTIFICIAL SUNLIGHT

The use of heliotherapy for tuberculosis in general and for laryngeal tuberculosis in particular is not new. The healing power of the sun was surely known to ancient civilization. The Egyptians, Persians, Greeks and Romans employed sunlight for therapeutic purposes, and probably quoted the elder Pliny "Sol est remediorum maximum!" (The sun is the greatest remedy.)

During the long sleep of the dark ages, the expression of the use of heliotherapy was blotted out, and modern aspects of the subject are as recent as thirty years ago, when Poncet began to use sunlight in the treatment for tuberculosis. His work made little or no impression, and was left unnoticed. In the late nineties Finsen of Copenhagen made the world watch him when he employed the condensed rays of the sun in the treatment for lupus. From a scientific standpoint, he was the

founder of modern heliotherapy, and was also the first to find in the carbon arclight an excellent substitute for sunlight, which was especially effective in lupus.

In 1898, John W. Kime of Fort Dodge, Iowa, took up the subject of condensed sun rays in the treatment for pulmonary tuberculosis. Although he exhibited photographs and demonstrated a large sun reflector for the concentration of sunlight on the chest in pulmonary tuberculosis and on open wounds in surgical tuberculosis, and although his work was published in such well known periodicals as *The Journal of the American Medical Association* and the *New York Medical Journal,* he shared the fate of Poncet, for his observations were overlooked. His claims should be investigated, for he appears to be one of the American pioneers of heliotherapy in tuberculosis.

In 1902, Bernhard of St. Moritz was credited with using sunlight in the treatment of infected wounds and surgical tuberculosis. In 1903, Rollier of Leysin began his epoch-making experiments with sunlight. In 1904, Molgat of Nice devoted considerable attention to the results of heliotherapy in pulmonary tuberculosis.

However, it was Sorgo who first treated with reflected sunlight patients who had tuberculous lesions of the larynx, and he was able to demonstrate a number of successful cases twenty years ago.

Experiments conducted by Juengling, Hausser and Vahle to determine the exact power of the pigment formation of the individual segments of the spectrum coming from the mercury vapor light, the "quartz light," brought out the interesting fact that the power of pigment formation of the spectrum increases with the decrease of the wave lengths. But only up to a certain portion of the spectrum does it attain its maximum power of pigment formation, namely, at 300 millimicrons, which is known as the inner ultraviolet. At the outer ultraviolet, although the wave lengths are still shorter, this power decreases. Mercury quartz light yields a great amount of these short rays belonging to the outer ultraviolet. This observation is very important, as the reader will observe later.

Strandberg, who worked in the Finsen institute and used an ordinary carbon arclight in the treatment for laryngeal tuberculosis, reported relatively favorable results. Strandberg's encouraging results impressed Professor Hajek, and he delegated his assistant, Emil Wessely, to investigate the value of the carbon arclight with regard to its use in the treatment for tuberculosis of the upper air passages.

It was Wessely's intention not only to test the favorable results obtained by Strandberg, but to investigate the physiology and biology of sunlight and its substitutes with a view to produce an artificial source of light resembling sunlight. Since it is known empirically and theoretically that the maximum amount of light energy causing erythema and

pigmentation is due to the rays having a wave length of 300 millimicrons, and that these are located at the outer or blue end of the spectrum, namely, the ultraviolet end, Wessely began to experiment with these rays to the exclusion of all others.

The plan was to imitate the blue end of the sun's spectrum as closely as possible by the employment of a tremendous light energy and an apparatus that would allow this energy to strike unaltered at a desired spot.

The carbon arclight yields rays closely resembling sunlight, but the ordinary arclight in itself is not the best imitation of sunlight. With the addition of a certain substance, metallic salt, to the carbon bars, the blue end of the spectrum becomes complete. Such impregnated carbons are now manufactured by the optical concern, the C. P. Goerz Company of Vienna. These impregnated carbons yield rays that have a wave length of 290 millimicrons, embracing the entire ultraviolet of high altitude sun at zenith. The spectral analyses of these carbons were made by Professor Hascheck of Vienna.

In order to destroy tuberculous tissue, powerful and concentrated light is necessary, light of the inner ultraviolet having a wave length of about 300 millimicrons. This has been scientifically accomplished in the Wessely radiation machine (modified carbon arclight). By a specially designed Goerz system the superficial brightness is doubled, which also shortens the time of exposure to irradiation.

The following requirements are necessary for this artificial light to be of value: It must be a chemically active, cool light of a suitable photochemical intensity. It must be concentrated so as to reduce the time of exposure to a minimum and at the same time to create an optimal therapeutic effect. These requirements are all fulfilled by the Wessely machine, which is an arclight burning with specially constructed and impregnated carbons according to the Goerz system, by which the greatest concentration of rays is thrown to one side and made to converge into a cone through the medium of a quartz optic. The rays of heat are absorbed by a water jacket connected with the quartz optic. In other words, this machine is a carbon arclight, water-cooled, quartz lamp.

Only one patient at a time can be treated with this lamp, either directly by means of the Seiffert direct universal laryngoscope or indirectly by means of an all metal laryngeal mirror. Patients in good general condition or otherwise favorable subjects without involvement of the epiglottis are irradiated directly. Patients so treated have the benefit of the rays striking the lesion, thus shortening the period of treatment considerably. Frail persons, patients with advanced pulmonary lesions or patients who are intolerant and too sensitive and

Fig. 1.—Wessely radiation machine (modified carbon arclight), which is used in the treatment for laryngeal tuberculosis; it is especially designed to double the superficial brightness and to shorten the time of exposure. *a, b, c,* indicate crank-wheels, which permit every desired position, wheeling about, tilting and at the same time, fixation; *d,* condensor and water cooler; *e,* mirror and screen (magnifying mirror for self-observation); *f,* automatic regulation of electrodes, and *g,* observation window.

have lesions of the epiglottis are treated indirectly in the sitting posture by means of an all metal laryngeal mirror that is held suspended by a special contrivance attached to the teeth. Steel mirrors are utilized, because they reflect about 44 per cent of the valuable therapeutic rays into the larynx, while the ordinary glass mirrors absorb these rays and

Fig. 2.—Direct method of irradiation. The patient is suspended by means of the Seiffert direct universal laryngoscope, with the rays striking the lesion directly.

Fig. 3.—Patient looking at his own larynx. Indirect method of irradiation. The patient sits in front of a perforated magnifying mirror, and, with the laryngeal mirror suspended, he can regulate the rays of light directly on a given lesion.

reflect only about 9 per cent. This method is not as effective as the direct method, but it is more comfortable. The patient sits in front of a large mirror, which is centrally perforated like a head mirror, and can observe his own larynx. In this manner he is able to regulate the laryngeal mirror whenever the rays do not strike at a desired area.

During my stay in Vienna in October, 1924, I obtained permission from Professor Hajek and Dr. Wessely to examine and treat patients in the phototherapy department. What I saw made a profound impression on me. Superficial tuberculous ulcers heal rapidly under this form of treatment, leaving at first a small depression, which soon fills out to an even surface. Granulations become smaller and shallower, and one can distinctly differentiate the irradiated area from the nonirradiated one. Massive infiltrations of the posterior laryngeal wall change their shape and become smaller. Finally, there remains a pale, transverse elevation which on phonation is thrown into several folds.

The disappearance of dysphagia and greater roominess in the throat are the first favorable signs that the patient experiences, at times before any change in the appearance of the lesion is even noticeable. Of course, patients with extensive tuberculous destruction of the larynx—destruction beyond repair—may be influenced only symptomatically, and some patients, especially those with marked and progressive pulmonary lesions, may not be favorably influenced.

At any rate, this form of treatment gives results that hitherto have not been attained by any other form of treatment. The ability to swallow food and to drink comfortably gives these unfortunate persons a chance to fight the primary pulmonary condition. Ambulatory and afebrile patients and those without progressive pulmonary lesions are best suited for this kind of treatment.

The results obtained by Wessely in Vienna prompted me to import this machine to the United States in 1925. One year later, the trustees of the Beth Israel Hospital, on the advice of Dr. Kopetzky, installed this machine in the outpatient department and organized a special clinic for laryngeal tuberculosis in conjunction with the bronchoscopic clinic.

OBSERVATIONS AND EXPERIENCES WITH THE WESSELY MACHINE

Before treatment is begun, the patient's condition is determined carefully. A thorough examination of the chest, both physical and roentgenologic, is made; the sputum is searched for tubercle bacilli, and the blood is examined to determine the relative rapidity of the sinking of the erythrocytes (Fahreus). The results of these examinations are repeated on several occasions and compared. Thus an index is obtained by which the status of the patient may be determined more or less accurately from time to time.

Of course, the best results are achieved in patients with good resistance and relatively good power of regeneration. Such patients are those with mild pulmonary lesions, which either become arrested or show slight progression. These forms are usually afebrile or subfebrile and yield either normal or slightly elevated results with the sedimentation

tests (Fahreus). So far as circumscribed processes are concerned, in a certain number of cases the use of the cautery or in tuberculomas actual surgical removal may bring about healing much sooner. But one must also remember that in certain localities, such as in the anterior commissure where a matting of the cords may take place, and in the region of the processus vocalis where perichondritis and ankylosis may result, these measures are dangerous. In such localities the treatment with artificial sunlight is safe and gives the best results, though it may take from three to six months to accomplish.

While in the great majority of cases the local treatment with light has no effect on the primary pulmonary condition, in a great number of cases it was observed that with the healing of the local lesion, there was general well being of the patients; the fever slowly subsided, and the pulmonary condition improved. On the other hand, cases were seen in which the pulmonary condition remained the same or became worse, and yet healing and cicatrization were brought about in the tuberculous lesions of the larynx. Case 5 illustrates this point clearly. In such cases, it takes months to bring about healing, but it is accomplished, nevertheless.

However, in many febrile cases, local healing is never accomplished. While the lesions remain stationary, the pain disappears. Much has been written about the analgesic action of sunlight, and its blessing has long been known. There are cases in which, because of low resistance and rapid pulmonary involvement, light has no effect, as I have already mentioned in this article and in a previous article on the subject.

I treated seventy-four patients both in private practice and at the bronchoscopic clinic and found that laryngeal tuberculosis is somewhat more common in males. In my series of cases the disease occurred most frequently in Italians, especially in young Italian girls between 17 and 22 years of age. The age of the youngest patient was 16 and that of the oldest, 73. The most frequent sites of the lesion were the interarytenoid space and the arytenoid area which showed ulcers, tuberculomas or pear-shaped infiltrations. The next most common locality was the epiglottis; here also the condition appeared in the form of an ulceration or infiltration; the least frequently involved were the vestibule of the larynx and the cords. Last but not least, there was the malignant type of tuberculosis that is described as a diffuse infiltration of the entire circumference of the larynx. In this form the swelling and infiltration are not limited to the mucous membrane alone, but involve the deeper structures as well and give rise to a diffuse tuberculous perichondritis which, if the patient lives long enough, soon collapses and is destroyed by a process of caseation necrosis. The resulting ulcerations are extensive and respond to therapy with light only when the patient's resistance is good and his pulmonary condition stationary. But in most

cases in which there is a diffuse tuberculous perichondritis, a fulminating progressive pulmonary involvement occurs which shatters the resistance of the victim and soon brings about his death.

During the course of treatment, the patient's general condition is examined from time to time, and febrile patients and those showing progressive pulmonary involvement or loss of weight and strength are put to bed for weeks at a time as soon as the agonizing dysphagia has disappeared. Complete rest in bed, fresh air and good food are the best therapeutic trio in all forms of progressive tuberculosis.

As to the special value of a high and dry mountainous climate in pulmonary tuberculosis, I agree with Fishberg that in many cases it tends only to break up the home, as many of the sufferers are better off where they can get plenty of rest and good food and not be obliged to shift for themselves. Everything depends on the course of the disease. Febrile cases with rapidly progressive lesions usually have a fatal termination, no matter where the patients are sent or how soon they are sent. On the other hand, patients with the slowly progressive, chronic type of case with or without occasional elevations of temperature will linger for years, or their processes may become arrested altogether even in large cities under favorable homelike surroundings. But in laryngeal tuberculosis, particularly in the ulcerative types, residence in the country is of no help. What benefit is the fresh air to the sufferer when he or she is unable to swallow? Such patients are much better off in the city or at a sanatorium devoted to the treatment of patients with laryngeal tuberculosis.

REPORT OF CASES

Case 1.—C. St. J., aged 37, was referred from the tuberculosis clinic of the department of health on Dec. 14, 1925, with the complaint of hoarseness for the past five months and slight pain on swallowing for the past six weeks. He had had pulmonary tuberculosis for several years with cough and expectoration.

Examination showed an infiltration of both ventricular bands, partly covering the vocal cords, which did not approximate centrally. The interarytenoid space was filled with tuberculous granulation tissue. Bacilli were found in the sputum and the Fahreus test showed a sinking speed of 15 per cent in forty-five minutes.

The first treatment was administered on Dec. 22, 1925, and on every other day thereafter until seventeen treatments had been given. All exposures were direct via the Seiffert direct universal laryngoscope, with a chest support, and they lasted for from eight to ten minutes. Following the second exposure the pain on swallowing disappeared entirely, and after fifteen treatments the voice became normal. The ventricular bands assumed a normal aspect, and the masses in the interarytenoid space gradually faded away. The patient was discharged on Feb. 18, 1926, and he left to find work out of town.

Case 2.—S. K., aged 53, was referred from the Workmen's Circle Sanatorium through Dr. Halpern on Nov. 11, 1925, with the following complaints: difficulty in breathing, pain on swallowing, a choking sensation in the throat, cough and expectoration. The pulmonary status was bilateral advanced pulmonary tuberculosis with cavitation.

Laryngoscopy revealed tremendous swelling of the epiglottis, arytenoid folds and arytenoid region, interarytenoid space and vestibule. The right cord was not visible; the left was partially so. The chink of the glottis was narrowed, so that there was interference with respiration. Several deep ulcers were present on the laryngeal surface of the epiglottis, arytenoid region and interarytenoid space. The entire picture was that of an extensive tuberculous perichondritis.

The sinking speed of the erythrocytes (Fahreus) was 48 per cent in forty-five minutes, and the sputum was loaded with tubercle bacilli. The evening temperature ranged between 99 and 100 F., and the pulse rate was 90.

Treatment was begun on Nov. 11, 1925, and was continued on alternating days for fifty-six exposures. All irradiations were indirect, an all steel laryngeal mirror

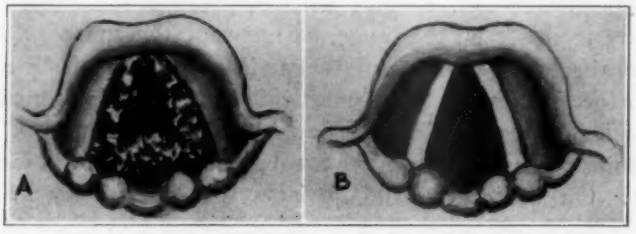

Fig. 4 (case 1).—*A* shows infiltrations of ventricular bands; interarytenoid space filled with tuberculous granulation tissue. *B* shows larynx entirely healed.

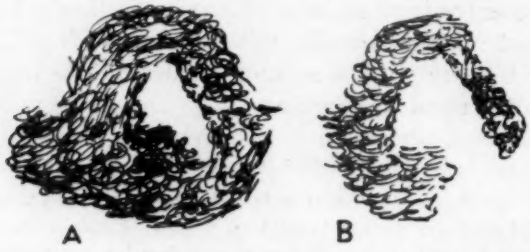

Fig. 5 (case 2).—*A* shows swelling of epiglottis, arytenoid folds, interarytenoid space and ventricle. Right cord is not visible; left cord is partly so. *B*, three months after irradiation. Both cords are visible. Most of the infiltration is gone.

being used; the duration of exposure at the beginning was five minutes and was increased to ten minutes. After six irradiations the pain was lessened, the respiration was less labored, and the patient volunteered the information that there was greater roominess in his throat. On Jan. 6, 1926, after the twenty-fourth treatment, the patient was entirely free from pain; he could swallow food and drink with ease, and he breathed freely.

Laryngoscopy performed on January 20 revealed no ulceration, lessened tumefaction, partial collapse of the epiglottis or distortion and disfiguration of the interior of the larynx. The Fahreus test gave a sinking speed of 54 per cent in forty-five minutes. The pulse rate and the temperature remained the same. Before the forty-sixth treatment, the patient complained of increasing weakness, gradual loss of weight and strength and poor appetite; yet the little food he ate was

swallowed without effort or pain. The sedimentation test on March 30 gave a reading of 60 per cent in forty-five minutes. Three days later, after the fifty-sixth exposure, treatment was discontinued, and the patient was ordered to bed. He died two months later from pulmonary tuberculosis. His family informed me that he could eat and swallow food painlessly and was free from laryngeal symptoms to the end.

CASE 3.—Miss L. R., aged 42, was referred from Arizona, where she had been confined to a sanatorium for treatment for pulmonary tuberculosis. At the first consultation she complained of increasing hoarseness, cough and expectoration. The pulmonary condition dated back many years; she was familiar with the nomenclature of tuberculosis, and offered information as to how many cavities she had and where they were located. Hoarseness had begun gradually in September, 1925.

Laryngoscopy on April 5, 1926, revealed swollen and congested cords, slight infiltration of the arytenoid region and granular masses in the interarytenoid space. The pulse rate was 96 and the evening temperature was 99 F. Respiration was asthmatic; she was somewhat cyanosed, and the sputum was bulky, tenacious and bacillary. The Fahreus test gave a sinking speed of 24 per cent in forty-five minutes.

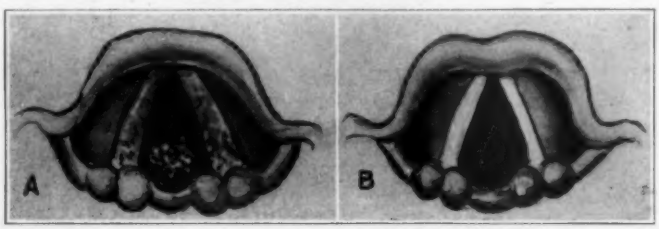

Fig. 6 (case 3).—*A* shows swollen and congested cords, slight infiltration of arytenoid region and granular masses in the interarytenoid space; *B*, larynx completely healed after eighteen exposures.

The first irradiation was administered on April 5, 1926; indirect exposures of ten minutes every session were given on alternating days. A total of eighteen exposures was administered, making a combined total of three hours of actual irradiation. During the course of treatment, the voice gradually cleared and soon assumed a normal tone. The last I heard from her was that she had had a thoracoplasty recently at Mount Sinai Hospital to put at rest her diseased lung. The larynx, however, was healed.

CASE 4.—Dorothy R., aged 18, came to the hospital on Feb. 18, 1926, complaining of hoarseness for the past three months and cough and expectoration for the past five weeks. Ten days prior to her first visit she had begun to experience slight pain on swallowing, which became progressively worse as time went on and at the time of the examination was unbearable. She had been only a few months in this country, having emigrated from Russia, where she had to go through a severe ordeal in her early childhood during the war and had been forced to subsist for months at a time on turnips and various other herbs.

Examination revealed an extensive infiltration of the entire interior and circumference of the larynx with the appearance of a waxy, rigid mass involving the deeper layers. The entire picture was that of an acute, fulminating, tuberculous perichondritis. Roentgen examination of the chest, reported by Dr. Joseph

Diamond, revealed an extensive peribronchial infiltration, extending to the parenchyma of both lungs. These observations were indicative of an acute fulminating form of pulmonary tuberculosis.

The entire course consisted of thirty-four exposures between the dates of Feb. 19, 1926, and April 17, each of from eight to ten minutes' duration. After the fourth exposure the pain lessened, and after the tenth it disappeared. The patient could swallow food and could drink without effort, but objectively the appearance of the larynx remained the same. In view of the fact that the subjective laryngeal symptoms subsided without any appreciable change in the pathologic structure of the larynx, and in view of the fact that the sinking speed of

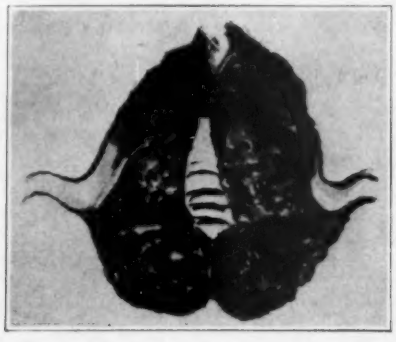

Fig. 7 (case 4).—Extensive infiltration of entire interior and circumference of larynx. Massive tuberculous perichondritis.

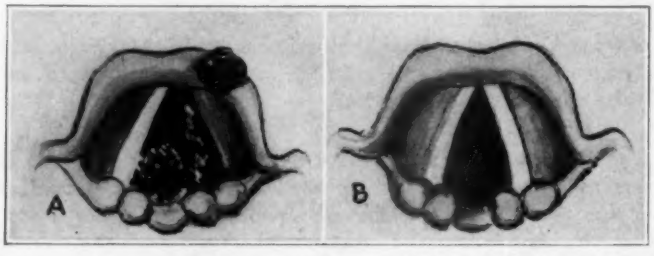

Fig. 8 (case 5).—*A* shows ulcer of the left upper margin of the epiglottis, a serrated and thickened left vocal cord and tuberculous granulation tissue in the interarytenoid space; *B*, appearance of larynx after nineteen irradiations.

the erythrocytes became faster as time went on, it was decided to suspend treatment and give the patient complete rest. For the next five months I was in communication with her parents, who assured me that she was able to swallow food and to drink painlessly. The end came while the patient was sitting comfortably and looking through the window, having had no complaints.

Case 5.—Mrs. L. S., aged 34, was referred to us from the tuberculosis clinic of the department of health on Dec. 30, 1925, her chief complaint being pain in the throat for the past three months, especially on swallowing. She also complained of cough and expectoration, loss of weight and strength and exhausting night sweats.

Laryngoscopic examination revealed an ulcer beginning at the upper margin of the epiglottis and extending to the middle of its laryngeal surface. It was covered with a dirty, gray, necrotic membrane, and surrounding it was an inflamed, elevated and serrated margin. The sputum was bacillary, the Fahreus test gave a sinking speed of 30 per cent in forty-five minutes, and the roentgenogram showed an extensive infiltration of both pulmonary fields.

The first treatment was administered on December 30, and lasted for five minutes. All of the other exposures were given for eight minutes on alternating days for a total of nineteen treatments. After the fourth exposure, the pain gradually lessened, and soon no mention was made of it. The necrotic membrane gradually cleared, and here and there healthy islands of granulation tissue were seen budding from the ulcerated surface. Finally the entire ulcerated area was covered with healthy granulations, which during the course of treatment cicatrized and eventually healed. In spite of all that, the patient made little progress. Although she could swallow food painlessly, her appetite was poor; she did not gain weight or strength, her night sweats and the cough and expectoration continued. She was sent to an institution, where she was last heard from in May, 1928, after repeated attacks of hemoptysis; she was still courageously fighting the pulmonary tuberculosis.

Case 6.—S. C., aged 28, was referred to me by Dr. I. Russel Kuhn on Aug. 24, 1926. His complaint was hoarseness for the past two months and pain in the throat, especially on swallowing, for past four weeks. This occurred while he was staying in the country on account of pulmonary tuberculosis. He was not febrile, nor did he have any other subjective symptoms referable to his pulmonary condition. The pain in the throat, however, was severe, radiating to the ears every time he swallowed.

Laryngoscopic examination revealed a uniform swelling of the epiglottis with several ulcers on the upper margin, infiltration of the aryepiglottic folds and arytenoids, swollen and congested cords which did not properly coaptate and ulceration of the interarytenoid space. An oval ulcer about ¾ inch (19 mm.) in length and ½ inch (12 mm.) in breadth, surrounded by an elevated irregular margin and covered by a dirty gray exudate, was present on the right side of the posterior pharyngeal wall. The pulse rate was 80; the Fahreus test gave a sinking speed of 18 per cent in forty-five minutes, and the sputum was rich in tubercle bacilli. Roentgen examination of the chest revealed extensive infiltration of both pulmonary fields.

Treatments were begun on Nov. 13, 1926, three weeks after his first consultation, as the patient was undecided about taking the treatments. He was now in a wretched condition. But once treatment was started, it was given uninterruptedly three times a week until June 24, 1927. During this time the patient gained 40 pounds (18.1 Kg.) in weight; the pain in the throat had entirely disappeared, but the hoarseness still persisted, although to a lesser degree. Laryngologically, the picture was now entirely different. All of the ulcers were healed, the swelling of the epiglottis and of the arytenoid folds was greatly diminished, and the infiltration of the arytenoid region and the cords was markedly lessened. The entire larynx had assumed a more normal form, and the mucous membrane, its characteristic luster. The patient then left for the country, where he rested for three months, returning in excellent health.

Case 7.—M. J., aged 22, was referred from the tuberculosis clinic of the health department on June 8, 1927, with a history of having suffered from pulmonary tuberculosis for four years. During the past few weeks he had begun to lose weight more rapidly; he had pain on swallowing and was slightly hoarse.

Examination of the larynx revealed a deep ulcer in the middle of the epiglottis, which extended deeply downward and inward to about its center. The rest of the epiglottis was infiltrated, and there was some infiltration at both arytenoid regions. At this time, his weight was 106 pounds (48 Kg.), the Fahreus test gave a sinking speed of 21 per cent, the temperature was 100 F., and the sputum was bacillary.

Dr. Stivelman reported the following observations on examination of the chest: infiltration and multiple cavitation of the entire left lung, infiltration of the upper part of the right lung, a thickened pleura in the lower part of the left lung, the heart and the trachea pulled to the left. The roentgenograms corroborated these observations.

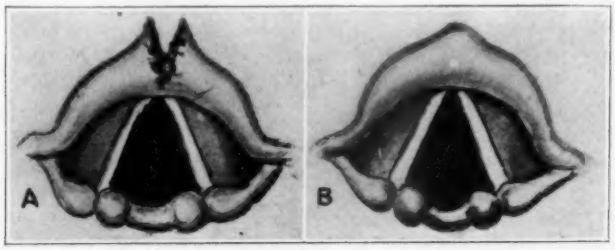

Fig. 9 (case 7).—*A* shows a deep ulcer in the middle of the epiglottis; *B*, the larynx four months later, practically healed.

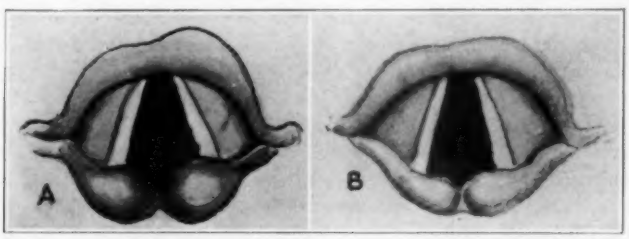

Fig. 10 (case 8).—*A* shows a pear-shaped swelling of the epiglottis and infiltrated arytenoids; *B*, appearance of larynx after seven months of treatment.

Irradiation was begun on June 9; the patient reported on June 11 that he was feeling better. Irradiation was given three times weekly for ten minutes at each exposure. On June 18, the patient reported a sharp pain in the left side of the throat; on June 28, he was free from pain. On August 18, the ulcer had practically healed, being almost covered by a pale-looking epithelium. The result of the Fahreus test was 35 per cent, and the weight was still 105 pounds. The patient was advised to go to a sanatorium for the condition of his chest and he was sent to Denver. From there the patient wrote: "I am feeling fine and have no trouble with my throat."

Case 8.—Miss M. E., aged 20, was referred from the tuberculosis clinic of the department of health on Nov. 22, 1927. In brief, her history was that until a year before, when she began to cough, she had been well. She visited the health department and was told that she was suffering from pulmonary tuberculosis. There was no loss of weight, night sweats or hemoptysis.

Laryngoscopic examination showed a pear-shaped swelling of the upper border of the epiglottis, tapering from right to left, an infiltrated left arytenoid and tumefaction of the interarytenoid space. The result of the Fahreus test was 5 per cent; the sputum gave a positive reaction.

Examination of the chest, as reported by Dr. Stivelman, showed infiltration and cavitation of the upper lobe of the right lung and infiltration scattered radially from the hilus of the left lung. The patient was irradiated from Nov. 26, 1927, to June 22, 1928, receiving ten-minute exposures three times a week; on the latter date the epiglottis was entirely healed, and the swelling of the arytenoid had subsided almost completely. The patient was sent for the summer to the country, and she returned to the clinic the last week in October with the larynx entirely healed. The sputum, however, still showed occasional tubercle bacilli.

Case 9.—Frances K., aged 20, was referred to the hospital from the tuberculosis clinic of the department of health on June 21, 1927, with the following history: She had had pneumonia eight months previously, and since then had lost 20 pounds (9 Kg.) in weight. Two months previously, she had become hoarse, and the hoarseness had gradually become worse.

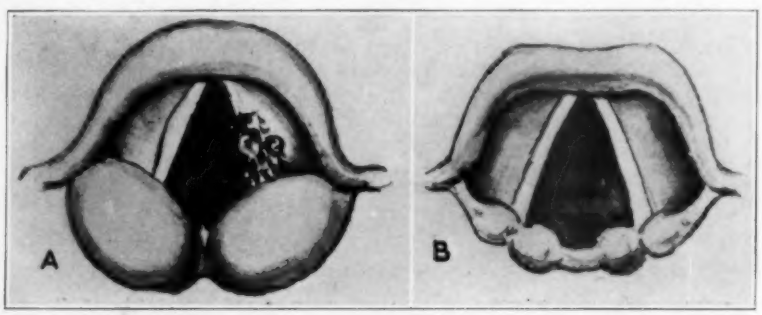

Fig. 11 (case 9).—*A* shows tuberculous granulation tissue in the posterior commissure. The posterior third of the left cord is ulcerated, with similar involvement of the posterior third of the left ventricular band. There is marked swelling of the arytenoids. *B*, after ten months of treatment, the larynx was entirely healed.

Laryngoscopic examination revealed: a collection of tuberculous granulation tissue in the posterior commissure, the posterior third of the left cord ulcerated and similar involvement of the posterior third of the left ventricular band. The motion of the left cord was somewhat limited. The Fahreus test showed 40 per cent of sedimentation.

Because the patient was free from pain on swallowing and because of her great debility, owing to the pulmonary condition (extensive involvement of both sides with cavitation), she was sent to a sanatorium. On Dec. 31, 1927, the patient returned, having gained 17 pounds (7.7 Kg.), and the sinking speed of the erythrocytes was now reduced to 35 per cent. The patient was irradiated three times a week, each exposure lasting for ten minutes. On April 5, 1928, the swelling of the arytenoid area had noticeably subsided; the granulation tissue of the posterior commissure had disappeared, and the left cord was entirely healed. On May 15, treatment was discontinued, and the patient was sent to spend the summer in the country.

CASE 10.—Joseph D., aged 29, was referred by Dr. Nathan D. Wolf on Feb. 25, 1928, with the complaint that he had had tuberculosis for several years, and that hoarseness had been present at times, but the last time had lasted longer (three weeks) and was getting worse. For the past two weeks there had been pain on swallowing, which in the past few days had been radiating to the ears, especially to the right ear. Shortness of breath, incessant cough with expectoration, loss of weight and strength and occasional night sweats were other subjective symptoms.

Laryngoscopic examination revealed an infiltrated and ulcerated epiglottis, ulcerations in the interarytenoid space, infiltrated arytenoids and swollen ventricular bands. The sputum gave a repeatedly positive reaction.

Irradiation was first administered on Feb. 25, 1928, and three times a week thereafter, with ten-minute exposures until twenty treatments had been given. The last exposure was given on May 31. After the fifth exposure, the pain in his throat eased, and it lessened as time went on, disappearing entirely on the twelfth exposure. While the ulcerations healed completely, the laryngeal infiltrations only shrunk to some extent. He had been gaining weight rapidly, and as he was free from his laryngeal symptoms, he was advised to spend the summer in the country.

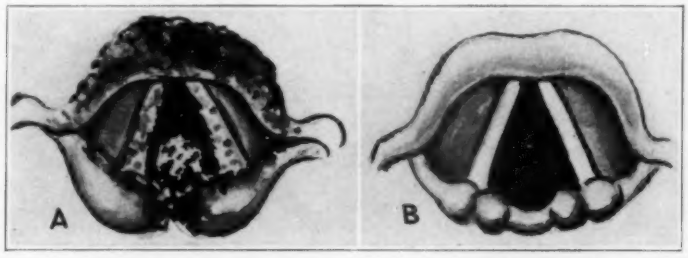

Fig. 12 (case 10).—*A* shows infiltrated and ulcerated epiglottis, ulcerations in interarytenoid space, infiltrated arytenoids and swollen ventricular bands. *B*, larynx entirely normal eight months later.

On his return in October, he looked and felt splendid. He is now being irradiated once a week so as to reduce still more the few remaining infiltrations.

CASE 11.—D. L., a married man, aged 39, was referred to me by Dr. Wilson of Saranac Lake on June 6, 1928. About two years prior to this date, frequent hemoptysis, cough and expectoration had begun. Hoarseness developed, becoming progressively worse and constant. He was sent to Saranac Lake, where he spent nineteen months and regained his weight and strength, but he did not regain his voice.

Laryngoscopic examination revealed uniform swelling and congestion of the mucous membrane of the epiglottis, cords and arytenoid region and infiltration of the ventricular bands. The mucous membrane of the interarytenoid space was covered with a dirty gray necrotic layer. Examination of the sputum gave repeatedly positive results.

Treatment was begun on June 12, 1928, with indirect exposures and was continued three times a week at the rate of ten minutes each until Aug. 8, 1928, when the mucous membrane of the epiglottis assumed a normal aspect. The patient was then given direct exposures once a week until October 12, when he was again treated in the indirect position, with only one irradiation a week. The infiltration of the arytenoid region and ventricular bands had disappeared; only the cords

were still somewhat thickened, but they met in perfect apposition on phonation. The patient had regained his voice fully, but complained of slight huskiness in the morning. At the time this paper was written, he was still under treatment.

CASE 12.—Samuel C., a robust man, aged 57, was referred by Dr. Herbert Adler, Feb. 11, 1928, with the chief complaint of hoarseness which had begun in May, 1927. Prior to that time he had had some pain in the throat, which had disappeared with the coming of the hoarseness. He had lost no weight and was afebrile, but shortness of breath and continuous cough gave him little or no rest.

Laryngoscopic examination revealed infiltration and congestion of the left vocal cord, with limited mobility. Examination of the sputum repeatedly gave positive results, and the result of the Fahreus test was 32 per cent. Dr. Adler's report was that the patient had a Wassermann reaction of 4 plus which, after a course of antisyphilitic treatment, was reduced to 1 plus. He also had a mild form of diabetes. Dr. Adler further reported that the condition of the chest was that of a bilateral chronic fibrous tuberculosis with cavities at both apexes.

The first irradiation was administered on Feb. 13, 1928, and the last May 29, 1928, during which time the patient received twenty-four exposures at the rate of ten minutes each. During the course of treatment his voice cleared up gradually, and soon was restored to normal. All of this time he was out on business, and he appeared to be in good health. However, I was informed recently by Dr. Adler that on June 17, he was seized with a severe attack of hemoptysis followed by high fever, which soon became irregular. On July 7, he left for Saranac Lake, where he died on August 5. His physician assured me that his voice was clear to the end, and that he did not suffer from any other laryngeal symptoms.

CASE 13.—Sophie P., a healthy-looking unmarried woman, aged 22, was referred to the hospital from the Bay Ridge Tuberculosis Clinic of the department of health on March 3, 1927, with the complaint that in April, 1926, she had suddenly become hoarse, and that this condition had continued. There was no pain but occasional cough and expectoration.

Laryngoscopic examination revealed a serrated left cord, which was much thickened, and which presented the appearance of a string of beads. The mucous membrane in the interarytenoid space was congested and redundant. The sputum showed bacilli and the Fahreus test showed a sinking speed of 10 per cent in forty-five minutes.

Roentgenographic examination of the chest, as reported by Dr. H. K. Taylor on April 1, 1927, showed tuberculous involvement of the upper lobe of the left lung and of the apex of the right lung.

Physical examination of the chest by Dr. Stivelman corroborated the roentgenographic observations.

The first treatment was given on March 3, 1927. Three times a week, indirect exposures were given for ten minutes each, and treatment was continued until June 28, 1927. On this date, a note by Dr. Hendleman on the chart stated: "Voice regained, cords less infiltrated and paler—advise one exposure a week." Irradiations now followed at weekly intervals from five to ten minutes at each session until April 27, 1928. On that date, the Fahreus test gave a sinking speed of 5 per cent in forty-five minutes. The patient's general condition was markedly improved, and she gained about 35 pounds (15.9 Kg.) in weight. She was then advised to take a rest and spend the summer in the country.

CASE 14.—Stephan H., aged 44, was referred from the tuberculosis clinic of the department of health on Aug. 11, 1927. His mother had died of tuberculosis at the age of 40 and one brother was ill with tuberculosis. The patient gave a history of having suffered from cough and expectoration for the past three years.

Hoarseness had begun eighteen months previously. Occasionally he felt pain in the throat which radiated to the left ear. Up to six months previously, he had consumed a considerable amount of alcohol.

Laryngoscopic examination demonstrated infiltrated ventricles, particularly the left, and that both cords were thickened and did not coaptate centrally. The interarytenoid space was involved with tuberculomas. Physical examination by Dr. Stivelman showed infiltration with cavitation of the upper right lobe and infiltration of the upper left lobe. A roentgenographic report by Dr. H. K. Taylor showed extensive exudative phthisis of the upper lobes of both lungs, with several small cavities. The sputum showed bacilli and 8 per cent of the sediment sank in the Fahreus test.

The first treatment was given on Aug. 11, 1927, by direct exposure which lasted eight minutes, and treatment was continued once a week thereafter until May 8, 1928. During that time the patient was indirectly irradiated eight times. All other exposures were direct. After the fifth treatment, pain was diminished, and the patient began to gain weight and strength and felt better generally. As he stopped coming to the clinic and moved away without leaving an address, I lost track of him.

Case 15.—Leonard D., aged 38, was referred from the tuberculosis clinic of the department of health through Dr. Silberman on Jan. 4, 1928, with a history

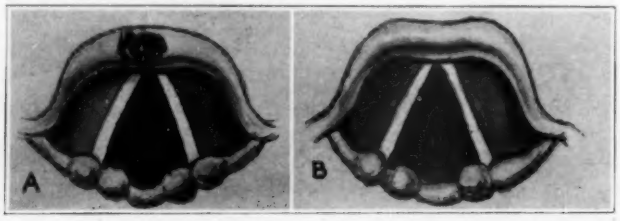

Fig. 13 (case 18).—*A* shows laryngeal surface of epiglottis thickened and ulcerated in the middle; *B*, epiglottis smooth and normal as it appeared three months later.

of having suffered from cough and expectoration and gradual loss of weight for the past year and pain in the throat, especially on swallowing, and slight hoarseness for the past eight months. The family history was unimportant, and there was no syphilitic history.

Physical examination by Dr. Stivelman showed infiltration and cavitation of the left upper pulmonary field and infiltration of the right apex. Roentgenograms corroborated the aforementioned observations. The sputum gave a positive reaction, and 10 per cent of the sediment sank in the Fahreus test.

Laryngoscopic examination showed the laryngeal surface of the epiglottis so thickened and ulcerated in the middle that it had the appearance as if a piece was bitten out.

Treatment was begun on January 4, and terminated on June 8, at the rate of three exposures a week for ten minutes by the indirect method. After the fourth irradiation, no pain was experienced on swallowing. The necrotic membrane that covered the ulcer had disappeared. After treatment for three months many islands of healthy granulation tissue were seen budding from the floor of the ulcer, which soon became epithelialized and healed. The patient gained about 20 pounds (9 Kg.) in weight and felt better generally. In this condition, he left to find work during the summer in the country.

CASE 16.—Viola M., aged 25, the wife of a physician, first began to complain of hoarseness on March 20, 1924. She had no fever; however, examination of the sputum gave a positive reaction, and the patient was about 10 pounds (4.5 Kg.) underweight. She was received at the Trudeau Sanatorium, and she remained there until November. When she returned she had gained 23 pounds (10.4 Kg.) in weight; her voice was considerably improved, and she was feeling very much better generally. Examination of the sputum still gave a positive reaction, and there was an occasional cough and clearing of the throat in the morning. On admission to the Trudeau Sanatorium, the upper lobe of the right lung showed slight involvement, and the right vocal cord was somewhat serrated. The patient was discharged as having a quiescent case, with the right vocal cord healed.

In January, 1928, dry, tickling sensations developed in her throat, with moderate dysphagia. Examination revealed a small punched-out ulcer of the right border of the epiglottis. Pulmonary observations confirmed by roentgenograms revealed bilateral apical fibrous tuberculosis with adhesions. There was no fever, but the patient was a few pounds underweight, and the sputum gave a positive reaction.

Treatments were begun on Feb. 14, 1928, and were continued three times a week thereafter until April 26. From then on two irradiations a week were given until July, when the patient was advised to suspend treatment and go to the country. All exposures were given via the indirect method and lasted ten minutes each. After the sixth exposure the discomfort in the throat and dysphagia disappeared. Gradually the ulcer shrank; the surrounding mucous membrane assumed once more its normal luster, and finally bridged over the area, leaving a shallow depression. She returned in October; she had maintained her usual weight and felt exceptionally well; she had no fever, but in occasional expectorations the sputum still gave a positive reaction. The ulcer healed, leaving a smooth depression. The mucous membrane of the upper border of the epiglottis was pink, though somewhat thickened over the former area of ulceration. At the time of writing, she was being irradiated only once a week.

COMMENT

Only two of the seventy-four patients treated did not respond to radiation. One was a man aged 73, and the other was a girl aged 20. In neither case was there subjective or objective improvement. Although the former reported for a time that he was feeling better, I am willing to discount that because of his anxiety to please and to cooperate with me, and because the improvement was only temporary. Both of these patients were very frail, and their condition was too far advanced; they had no regenerative powers. Fifty-nine of the other seventy-two patients showed complete healing of the laryngeal lesions with no subjective symptoms referable to the larynx; while the subjective laryngeal symptoms of the other thirteen patients improved, there was either partial or no healing of the tuberculous larynx. Of course, it should be emphasized that successful healing could have been produced in even a greater number had it been possible to administer irradiation to these thirteen patients in a sanatorium, where this mode of treatment should be carried out.

Although, to my knowledge, I am the first to import the Wessely Radiation Machine to the United States and actually to apply this form

of treatment to patients in this country, others before me have extolled its great healing power by word and pen. On May 13, 1925, at the Annual Meeting of the Medical Society of the State of New York, Dr. Roy S. Moore [2] of Syracuse read a paper on "The Treatment of Laryngeal Tuberculosis," in which he briefly and concisely discussed the the forty-nine patients treated by him at the Hajek clinic during his stay in Vienna. Also, in discussing a paper read by Frank Spencer [3] before the Section on Laryngology, Otology and Rhinology at the Seventy-Seventh Annual Session of the American Medical Association, at Dallas, Texas, in April, 1926, Dr. Joseph C. Beck of Chicago said:

> While I do not treat many cases of tuberculous laryngitis, the work of Wessely in the Vienna clinic in the use of the carbon light has revealed a method of treatment that I would not have believed if I had not tried it myself. Cases almost as bad as the one in which Dr. Hastings said he would not do surgery have been sent to me for injection of the superior laryngeal nerve. I gave treatment with the carbon lamp. The actual results were astonishing.

My two associates, Dr. Hendleman and Dr. Harris gave their best in aiding and assisting me in this laborious and highly specialized work. Miss Elizabeth Cutler helped us with excellent illustrative drawings.

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2. Moore, Roy S.: The Treatment of Laryngeal Tuberculosis, New York State J. Med. **26**:247 (March 15) 1926.

3. Spencer, Frank R.: Importance of Early Diagnosis and Treatment of Laryngeal Tuberculosis, J. A. M. A. **87**:216 (July 24) 1926.

GRADENIGO'S SYNDROME

WITH A CONSIDERATION OF "PETROSITIS" *

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In 1904 Gradenigo called attention to a definite clinical entity, namely, a suppurative otitis media complicated by involvement of the abducens and trigeminus nerves on the corresponding side. In the February, 1924, issue of the *Laryngoscope*, I reported a case with this triad; the patient recovered after a simple mastoidectomy. The incentive to the writing of this paper was an analysis of a second case with this syndrome, in which meningitis developed and the patient died; an autopsy was performed, and the temporal bone was removed with the corresponding side of the sella and cavernous sinus. This and two other cases stimulated anatomic studies of the petrous cells and adjacent structures at the apex. I made dissections, therefore, of the temporal bones of 5, 6 and 7 month fetuses, two full term infants and fifty adults.

One must consider the anatomy of the petrous bone and the structures at its apex in order to understand the routes of infection and the involvement of the fifth and sixth nerves. Gradenigo, in his report, quoted Dorello's description of the structures at the apex which form the abducens canal (now known as Dorello's canal). Vail [1] followed this description and presented sketches of the various stages of the dissection in the fetus. I also followed it in the dissection of the fetus, full term infant and adult; a composite drawing of the adult is shown in figure 1. The right side illustrates the relation of the nerves before penetrating the dura, and the left side the structures in and about Dorello's canal. It will be noted that the canal is formed as follows: above by the fibrous band (petrosphenoidal ligament) which is formed from the deepest layers of the tentorium, and below by the sphenoid and petrous bone. The canal is somewhat triangular or crescent-shaped and approximately 8 mm. wide and 4 mm. deep. The inferior petrosal sinus and the abducens nerve pass through this canal to enter the cavernous sinus. The third and fourth cranial nerves enter the cavernous sinus higher and farther forward. The abducens nerve is practically always in the outer border of the canal, which brings it in close relationship to the fifth nerve and its ganglion. This lies on a depression at the

* Submitted for publication, July 29, 1930.

* Read before the Section on Laryngology, Otology and Rhinology at the Eighty-First Annual Session of the American Medical Association, Detroit, June 25, 1930.

1. Vail, Harris H.: Anatomical Studies of Dorello's Canal, Laryngoscope **32**:569 (Aug.) 1922.

apex of the petrous bone. One is impressed in these dissections by the firmness of the dura overlying the trigeminal nerve. In fact, in stripping back the dura, fibers remain which are attached to the posterior clinoid process and to the ridge of bone that forms the roof of the internal auditory meatus (fig. 2). The ridge at times terminates in a spinous process (see illustrations of anatomic specimens). Even in the fetus the firmness of the dura simulates a ligament (fig. 3). The trigeminus nerve is consequently held snugly in a canal, bounded above by the dense dural fibers and below by the shallow "impressio trigmenis."

How does the inflammation get to the apex? I am convinced that this depends on the type and degree of pneumatization of the petrous bone. Ballance [2] gave the following explanation:

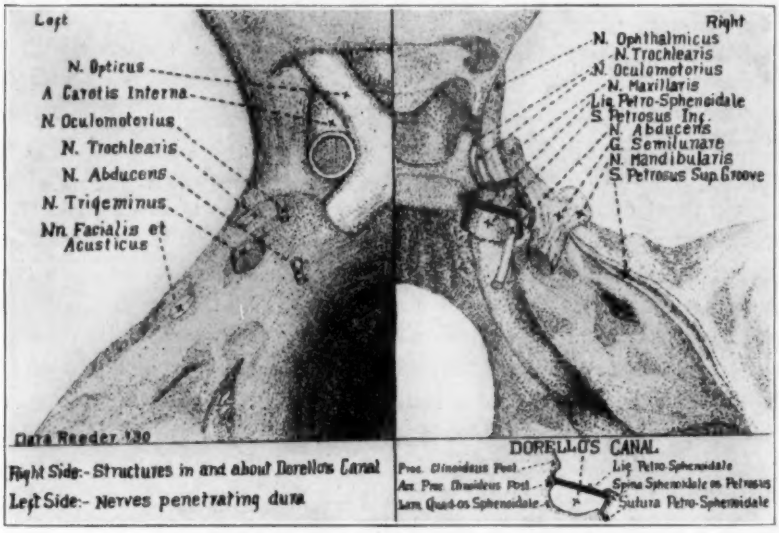

Fig. 1.—Structures at the apex of the petrous bone.

The too exclusive use of the term "mastoid cells" has possibly, to some extent, obscured the importance of other parts of the complex network of inter-communicating air-spaces which surround the tympanum. . . . The mucous membrane lining the whole of the complex space is a direct extension of that of the tympanum. The air-cells are in no way prevented by the sutures from extending, more or less widely and in continuity, from one bone to the neighboring bone.

It has become a convention to refer to the antrum as part of the mastoid, although embryologically and anatomically it is part of the petrous. In speaking of the petrous, therefore, it is necessary to refer only to that portion of the temporal bone which includes the labyrinth and the petrous tip.

2. Ballance, C. A.: Surgery of the Temporal Bone, New York, The Macmillan Company, 1920, p. 67.

The fundamental scheme of the pneumatization of the petrous bone is demonstrated in the dissection of the autopsy specimen already mentioned. Figure 4 shows the beginning of the dissection. A section was made at right angles to the long axis through the cochlea, and

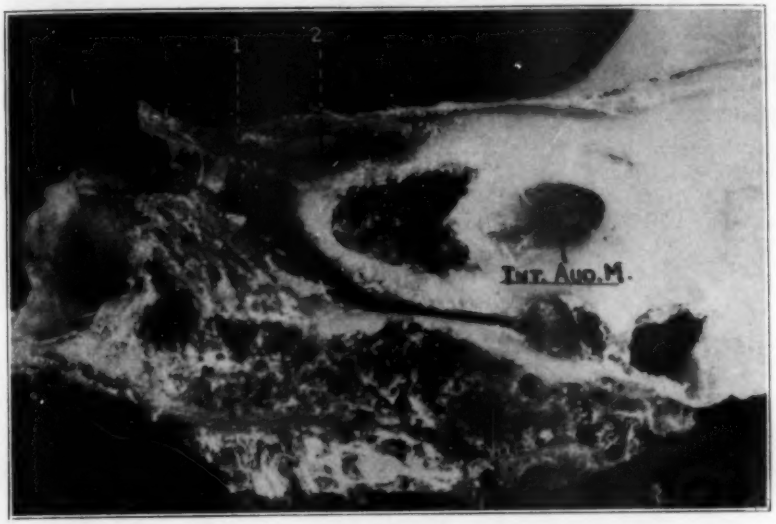

Fig. 2.—Anatomic specimen: 1, abducens (Dorello's) canal; 2, trigeminus canal; *Int. Aud. M.*, internal auditory meatus. Note the tense dural fibers.

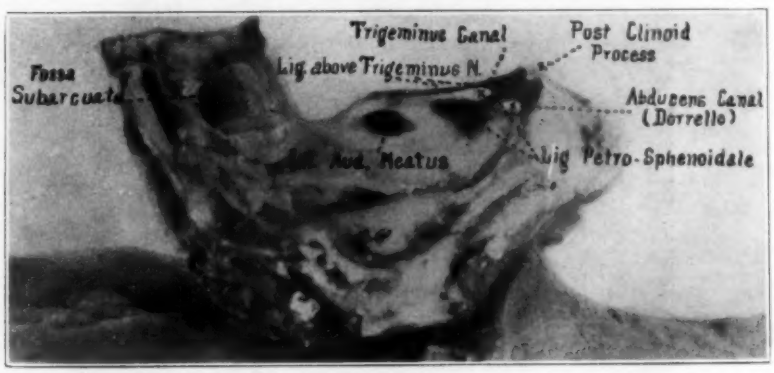

Fig. 3.—Petrous bone of a 6 month fetus.

figures 5 and 6 show the complete dissection. Figure 7 contains sketches of the sections. It will be noted that I described two routes:

1. The "antrum-epitympanic route": The cells extend from the antrum and epitympanic space (*A*) above the cochlea, and above and behind the superior semicircular canal (*B*); then behind, above and in

Fig. 4.—Posterior view of petrous bone in case 1 (Gradenigo's syndrome), at beginning of dissection.

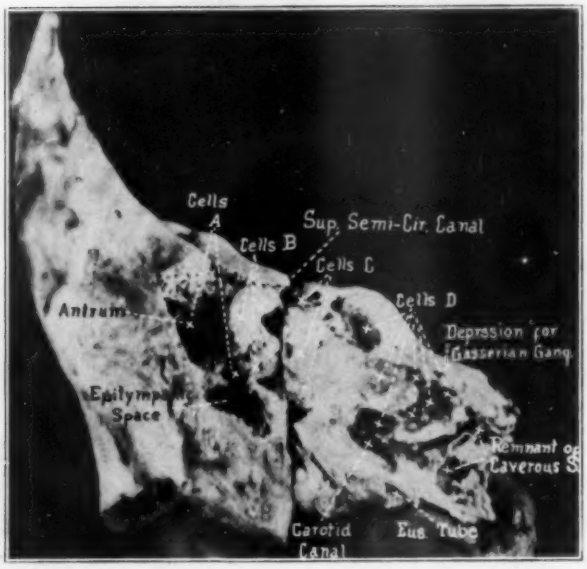

Fig. 5.—Anterior view of petrous bone in case 1.

Fig. 6.—Posterior view of petrous bone in case 1.

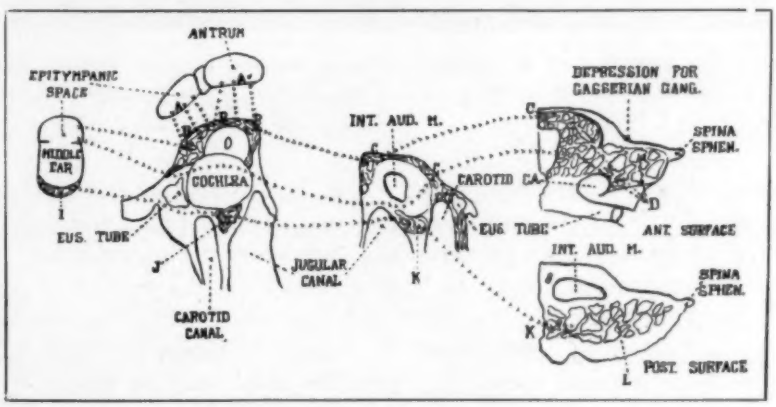

Fig. 7.—Sections in case 1: *A*, *B*, *C* and *D* indicate the antrum-epitympanic route, and *I*, *J*, *K* and *L* the hypotympanic route, of pneumatization of petrous bone.

front of the internal auditory meatus (*C*), and finally to the mass of cells under the tegmen of the anterior surface of the tip (*D*).

2. The "hypotympanic route": The cells extend from the hypotympanic space (*I*) below the cochlea (*J*); then below the internal auditory meatus (*K*), and finally to the mass of cells under the tegmen of the posterior surface of the tip (*L*). The two groups, *L* and *D*, meet immediately below the depression for the gasserian ganglion.

The dissections of other pneumatic petrous bones showed that the cells can always be traced to one of these two routes. The measurements of the two routes demonstrate that the distance to the tip is

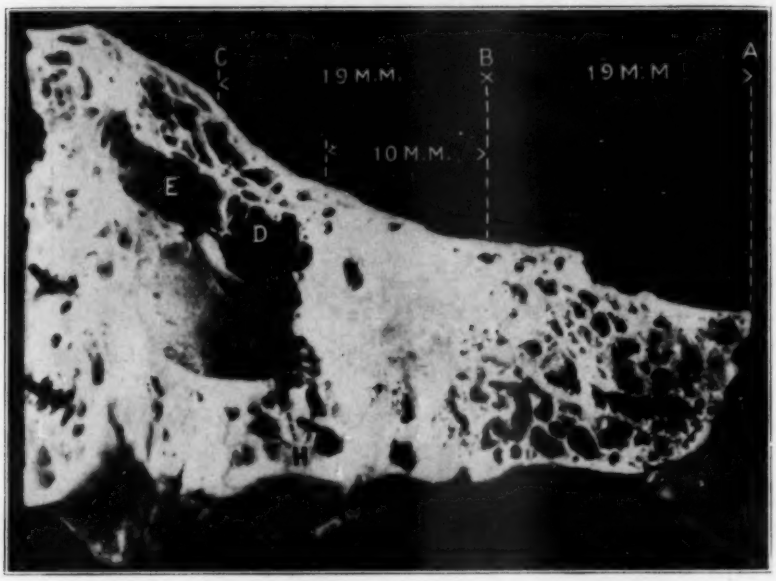

Fig. 8.—Anterior half of section in case 1; distance from epitympanic space (*D*) to tip, 10 mm.; from antrum (*E*) to tip, 19 mm.; from hypotympanic space (*H*) to tip, 7 mm.

shorter than one would suppose. This is due to the fact that the horizontal axis of the antrum-epitympanic space is practically parallel with the long axis of the petrous bone. The shortest distance, therefore, from the epitympanic space is 10 mm., and from the antrum 19 mm. The hypotympanic route is a direct path for only 7 mm. The measurements are shown in figures 8 and 9. Figure 10 shows the direct path of the hypotympanic route. It should be noted that the region commonly referred to as the petrous tip is 19 mm. in length.

From a clinical and surgical standpoint, the important thing to bear in mind is that the infection may extend from the hypotympanic space,

as well as from the antrum and epitympanic space. Perkins [3] has been quoted repeatedly since 1910, when he gave four possible routes. It is necessary, therefore, to analyze these routes. He gave the following: "(*a*) Sublabyrinthine route extending from the tympanum below the labyrinth and internal auditory meatus to the petrous tip." I call this the hypotympanic route because of its point of origin and because the term gives it a practical clinical significance. "(*b*) From the mastoid through the sub-arcuate fossa." This is a small portion of the antrum-epitympanic route. "(*c*) Via the carotid canal, access to which is obtained either by eroding the bone on the anterior tympanic wall, or through one of the carotico-tympanic plexuses." This is, of course,

Fig. 9.—Vertical section through anatomic specimen of adult petrous bone. Area between *A* (apex) and *B* (cochlea) is referred to as the petrous tip. Area between *B* and *C* (vertex of posterior semicircular canal) includes the entire bony labyrinth. *H*, hypotympanic space.

theoretically possible, but in either case the cavernous sinus would be affected before or at the same time as the trigeminus and abducens canals. "(*d*) Through a layer of cells extending along the eustachian tube, thus passing from the tympanum to the petrous tip." This is anatomically impossible, because the carotid canal is between the eustachian tube and the petrous tip. It is true that cells at times exist along the eustachian tube, but when they are above it they are from the epitympanic cells, and when below it they are from the hypo-

3. Perkins, C. E.: Ann. Otol., Rhin. & Laryng. **19**:692, 1910.

tympanic cells, while in either case only those at the orifice can communicate with the petrous tip.

The terms antrum-epitympanic route and hypotympanic route are further justified by a consideration of the embryologic development and architecture of the petrous bone. These may be briefly summarized as follows:

1. Ossification of the labyrinth commences in the fifth fetal month at five main centers (periotic); by the end of the sixth fetal month the individual centers are more or less fused.

2. Until the seventh fetal month the petrous bone consists only of the labyrinth, tympanum and antrum. There is no petrous tip; hence the cochlea is at the apex (fig. 11).

3. At birth the petrous tip consists of rudimentary projections from the cochlea. The carotid canal is still a groove, and the internal auditory

Fig. 10.—Probe shows direct path of hypotympanic route. 1, petrosphenoidal ligament (ossified); 2, Dorello's canal; 3, depression for gasserian ganglion; 4, internal auditory meatus.

meatus merely an orifice. The antrum-epitympanic space has its adult characteristics. There are no petrous cells, but the molding of the future antrum-epitympanic route is seen in the new bone deposit extending outward to the zygoma and inward above the labyrinth. This new bone deposit is brownish and soft in contrast to the dense white bone of the labyrinth; in fact, it is the same type of bone that forms the future mastoid process.

4. Siebenmann [4] and recently Tremble [5] have shown that at birth the labyrinth is practically the same size as in the adult, about 19 mm.

4. Siebenmann: Korrosionsanatomie, 1897.

5. Tremble, G. E.: Bony Labyrinth of New-Born Infant and of Adult, Arch. Otolaryng. **9**:175 (Feb.) 1929.

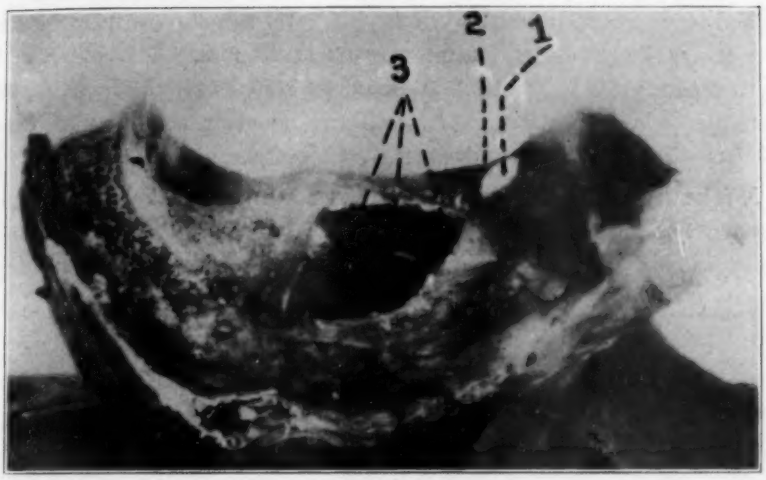

Fig. 11.—Anterior view of petrous bone in a 7 month fetus, showing that the cochlea (3) forms the apex of the petrous bone. 1, Dorello's canal; 2, petrosphenoidal ligament.

Fig. 12.—Anatomic specimen with a well developed and pneumatic mastoid.

The adult type of petrous bone, therefore, is the result of the molding of osseous deposits and structures about the fetal labyrinth, and it is reached between the fourth and fifth year. My measurements show that the average length of the adult petrous is 38 mm. The increase in length obviously is in the tip. Alexander [6] stated that the bone deposit above the labyrinth varies: Around the superior semicircular canal it is from 2 to 5 mm., and above the cochlea from 3 to 7 mm.

5. The hypotympanic space also has adult characteristics at birth. The hypotympanic route of petrous cells is affected by the ossification of the adjacent carotid canal and jugular fossa below the labyrinth. The

Fig. 13.—Pneumatic petrous bone in specimen shown in figure 12 (antrum-eptiympanic route to apex).

osseous space between the canal, fossa and labyrinth is the channel to the tip.

6. From a histologic standpoint the petrous cell is the same as the mastoid cell: a cavity lined with a thin mucoperiosteum. The development is coincident with and analogous to the development of the mastoid cell. The pneumatization of the adult petrous bone depends on the amount and distribution of the bone deposit on the fetal labyrinth, as well as on the degree of transformation of this deposit into cells by the

6. Alexander, Gustav, in Denker and Kahler: Handbuch der Hals- Nasen-Ohrenheilkunde mit Einschluss der Grenzgebiete, Berlin, Julius Springer, 1926, p. 87.

penetrating activity of the mucous membrane from the tympanum. This transformation is a normal process and not the result of an inflammation.

7. The dissections of the adult petrous bones show three types of bone formation: (*a*) compact or sclerotic, (*b*) spongy or diploic and (*c*) cellular. In a single specimen one, two or all three types may be present. For example, in three of my dissections cells extended to the

Fig. 14.—Hypotympanic cells in specimen shown in figures 12 and 13. External auditory canal removed. Photographic enlargement shows mass of cells. Note oval and round windows in depth. The cells were traced to those beneath the tegmen of the posterior surface of the petrous bone (hypotympanic route).

extreme end of the tip through both routes. In two, large cells were traced from the antrum-epitympanic route along the roof of the internal auditory canal to a spongy mass at the tip; in these two the hypotympanic route had only a few cells below the cochlea. In one specimen, in which the jugular bulb was low, there were many large hypotympanic cells extending below the labyrinth and internal auditory canal, but the

Fig. 15.—Anatomic specimen with small sclerotic mastoid (*M*) and sclerotic petrous bone (*P*). Bony labyrinth removed. 1, internal auditory meatus; 2, carotid canal; 3, eustachian tube.

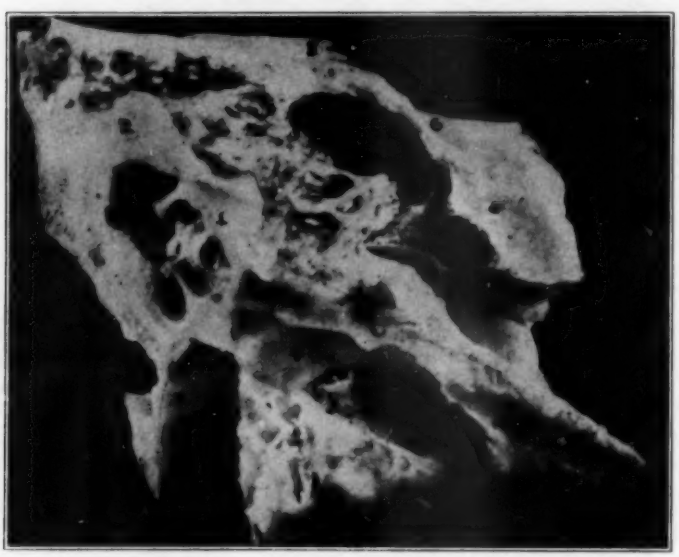

Fig. 16.—Vertical section (at a right angle to long axis of adult petrous bone. 1, cochlea; 2, internal auditory canal; 3, carotid canal; 4, eustachian tube; 5, spinous process; 6, depression for semilunar ganglion (antrum-epitympanic route to tip).

tip was spongy; the antrum epitympanic route had only a few cells over the cochlea while the rest of the anterior surface of the petrous was compact. As a rule, a specimen with a well developed mastoid had a well developed petrous, and one with a sclerotic mastoid had a sclerotic petrous. Figures 12 to 18 show the different types of pneumatization. In order to obtain statistics as to the frequency and extent of pneumatization of the petrous, several hundred dissections should be made. From the number that I have made I am convinced that the cells are frequently present, at least in some portion of the petrous. A review of the literature shows that relatively few postmortem studies of the petrous

Fig. 17.—Anterior surface of petrous bone; external auditory canal has been removed. 1, antrum-epitympanic route leading to cells in roof of internal auditory meatus (3); 2, superior semicircular canal; 4, spinous process extending from roof of interior auditory meatus; 5, depression for gasserian ganglion; 6, diploic tip.

cells have been made, not only in the Gradenigo syndrome, but in other inflammatory complications. Mangabeira Albernaz [7] showed a marked cellular development of the two routes terminating in a large tip cell (fig. 19). Ulrich [8] made microscopic sections of a specimen and found the cells leading to an abscess in the tip. Oppikofer [9] also made

7. Mangabeira Albernaz, Paula: Brasil-med. **41**:1149 (Oct. 29) 1927.
8. Ulrich: Ztschr. f. Hals-, Nasen- u. Ohrenh. **9**:404, 1924.
9. Oppikofer: Ztschr. f. Hals-, Nasen- u. Ohrenh. **21**:454, 1928.

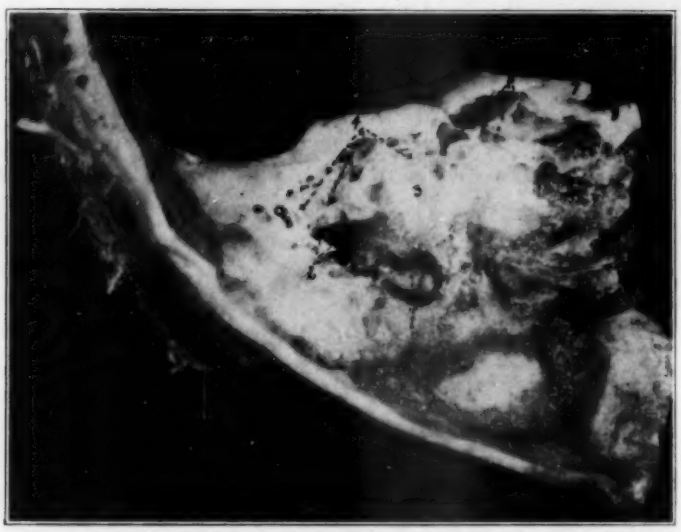

Fig. 18.—Anterior surface of petrous bone. 1, epitympanic space; 2, antrum; 3, superior semicircular canal; 4, cells leading to cells in roof of internal auditory canal (5) leading to diploic tip (6); 7 spinous process extending from roof of internal auditory canal; 8, depression for gasserian ganglion. In this type of pneumatization an inflammation could readily extend to and involve the trigeminus canal without affecting the abducens canal.

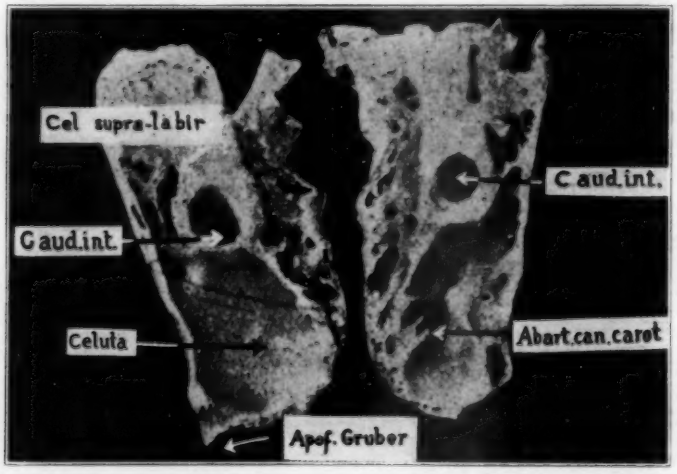

Fig. 19.—Note supra-labyrinthine cells (*Cel. supra-läbir.*) leading to large cells (*Celuta*) at tip (antrum-epitympanic route); also sublabyrinthine cells (hypotympanic route) leading to the cell. *C. aud. int.*, internal auditory canal; *Abart. can. carot.*, carotid canal; *Apof. Grüber*, apophysis of Grüber. This illustration was taken from Mangabeira Albernaz, P.: Brasil-med. **41**:1149 (Oct.) 1927.

microscopic sections of one and reported "marked pneumatization about the labyrinth to the median circumference of the internal auditory canal, and only a few small cells in the tip." In serial microscopic sections of an anatomic specimen made by Eckert-Möbius [10] there was a highly cellular hypotympanic route extending to the tip and no cells from the antrum-epitympanic route (fig. 20).

8. Because of the extreme variability of the pneumatization, no one case report, autopsy or even examination of the specimen can reveal the entire possible involvements of the petrous bone. Obviously, roentgen studies of the cells are of interest, but they are not diagnostic.

This study emphasizes the fact that a suppurative infection of the middle ear may be complicated by an involvement of the cells in the petrous bone analogous to a mastoiditis. An analysis of case reports shows that the petrous cells may be involved with little or no involvement of the mastoid. In the management, therefore, of an acute suppurative otitis media or mastoiditis, one must always consider the possibility of the presence and involvement of the petrous cells. With due apologies for suggesting another medical term, I believe that the word "petrositis" is justified, because it centers one's attention on this possible complication. Furthermore, it must be remembered that a petrositis may be present without obvious clinical signs. One sign suggestive of a petrositis is the presence, and especially the recurrence of, a profuse aural discharge, in the absence of definite involvement of the mastoid or during postoperative care after a mastoidectomy. The first definite sign to appear is pain in the areas supplied by the trigeminus nerve, most commonly in the temporoparietal area. The complete Gradenigo syndrome is relatively rare, but the occurrence of this pain in the course of a suppurative otitis media or mastoiditis is rather frequent. The reasons for this are that the cells seldom extend to the extreme end of the tip, and that the distance to the trigeminus canal is 10 mm. shorter than that to the abducens canal. In the cellular petrous bone the tegmen beneath the trigeminus canal is thin, and since the nerve is held snugly against the bone an inflammatory irritation readily occurs. When the cells extend to the extreme end of the tip or to a spongy bony mass in the tip, the invasion of the inflammation may cause edema with subsequent pressure within Dorello's canal. Depending on this pressure effect, there may be paresis or paralysis of the abducens nerve.

The point that I wish to emphasize is that a petrositis may be a complication of a suppurative infection of the middle ear. Too frequently in postoperative care after a simple mastoidectomy one is suddenly confronted with a rise in temperature, pulse rate, white blood

10. Eckert-Möbius, in Denker and Kahler: Handbuch der Hals-Nasen-Ohrenheilkunde mit Einschluss der Grenzgebiete, Berlin, Julius Springer, 1926, p. 242.

cell count, etc. An examination of the mastoid cavity reveals the wound in excellent condition. May not the cause of this acute exacerbation be an involvement of the petrous cells? This is excellently illus-

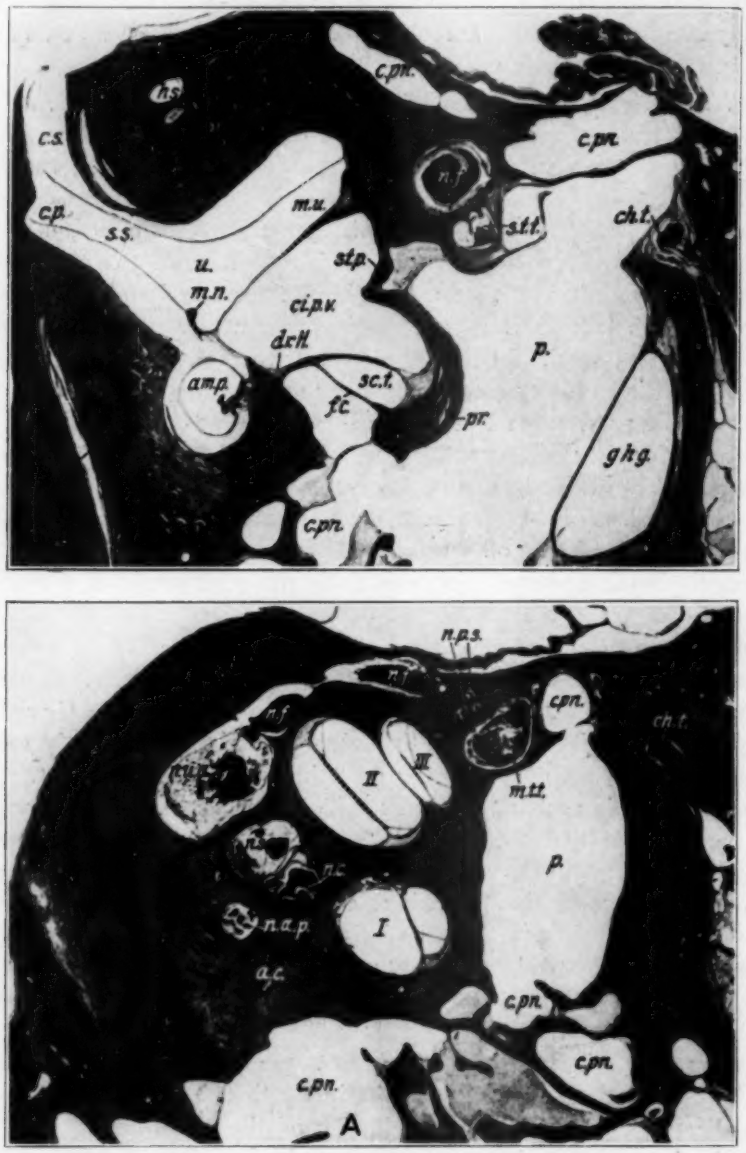

Fig. 20.—*A*, vertical section of anatomic specimen from tympanum to petrous tip (from Eckert-Möbius, in Handbuch der Hals-Nasen-Ohrenheilkunde mit Einschluss der Grenzgebiete); *p.*, tympanum; *pr.*, promontory; *c. pn.*, pneumatic cells; *I, II, III*, cochlea. Note hypotympanic route; hypotympanic cells lead to cells in the petrous tip (between carotid canal and internal auditory meatus).

trated in the report of a case by Dr. E. R. Roberts [11] (see appended case reports from the literature). I analyzed it as follows: Within seven days after the mastoidectomy the temperature was normal and

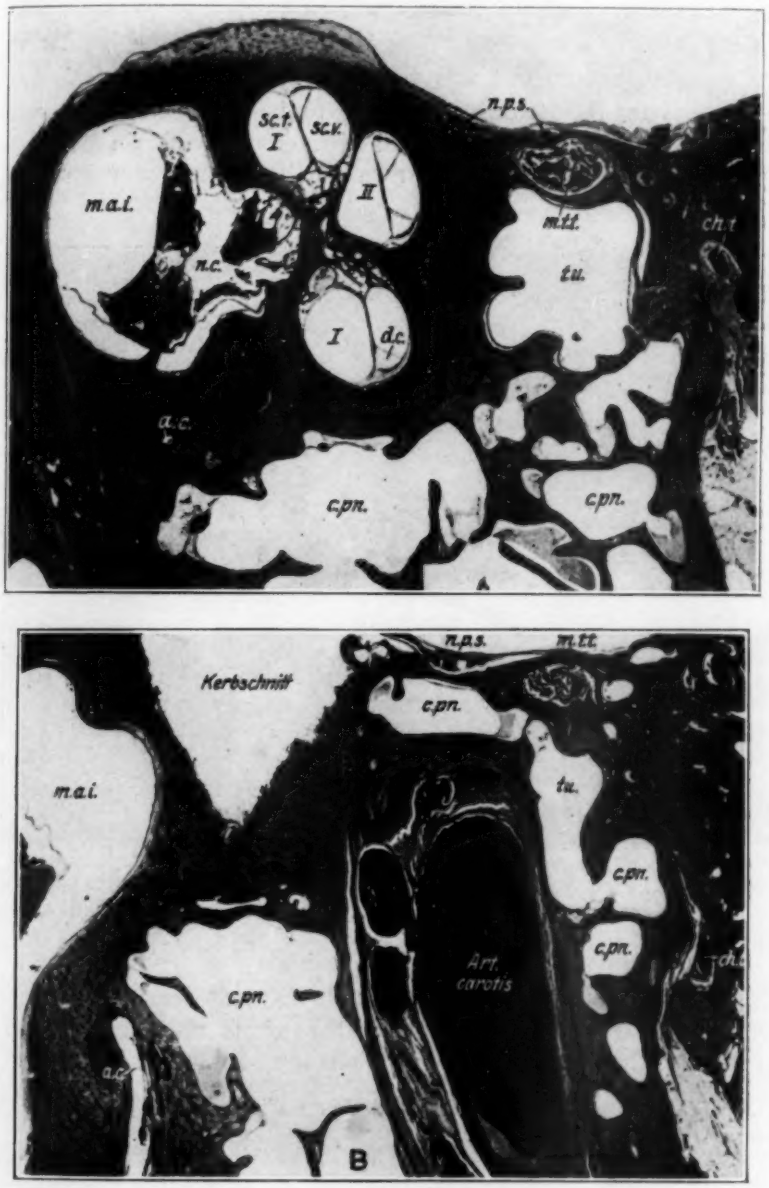

Fig. 20.—*B*, vertical section from Eckert-Möbius; *tu.*, eustachian tube; *m. a. i.*, internal auditory meatus.

11. Roberts, E. R.: Acute Otitic Temporosphenoidal Abscess, Arch. Otolaryng. **6**:420 (Nov.) 1927.

the patient's condition was good; the temperature then rose to 102 F., and there was pain about the temple and eye. (Here the petrositis began with irritation of the trigeminus.) Eight days later, the sixth nerve became involved, and there appeared a septic temperature which suggested a sinus thrombosis. The lateral sinus and the jugular vein were exposed, but no clot was found. (I believe that at this point the paralysis and the bacteremia were due to the involvement in Dorello's canal.) Later a temporosphenoidal abscess of the brain developed, and autopsy revealed an erosion through the tegmen of the superior surface near the apex.

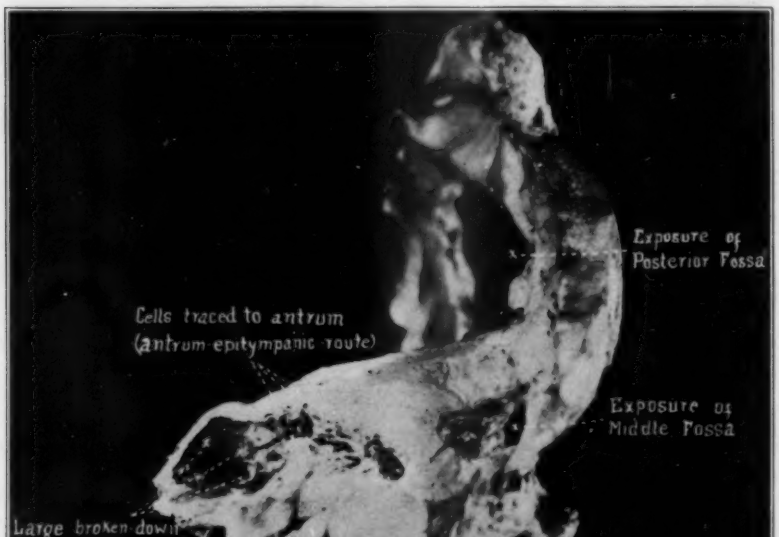

Fig. 21.—Autopsy specimen in case of petrositis without complete syndrome; subdural or brain abscess suspected; petrous cells found to be filled with purulent exudate.

My second case with Gradenigo's syndrome also illustrates the development of a petrositis and another of its complications. The essentials of the history and observations are as follows:

CASE 1.—A boy, aged 13, had acute otitis media on the right side, with a temperature of 102 F.

Second day: Spontaneous perforation, profuse discharge and a temperature of 99 F. Condition remained the same until the twelfth day.

Twelfth day: (Patient was seen for the first time.) Temperature 104 F.; all clinical signs of mastoiditis. A generous myringotomy was performed.

Thirteenth day: Mastoidectomy disclosed a large and pneumatic mastoid; many zygomatic cells found involved. The hemolytic streptococcus was isolated.

Eighteenth day: Good postoperative recovery; temperature from 99 to 100 F.; scanty aural discharge.

Nineteenth day: Temperature 104 F.; profuse aural discharge; mastoid wound explored and found in good condition.

Twenty-fourth day: Wound had been kept widely opened because temperature remained between 102 and 103 F.

Twenty-fifth day: Severe pain in right temporoparietal region.

Twenty-eighth day: Paralysis of abducens nerve; pain in area of trigeminus replaced by numbness over entire right side of face. Negative results from spinal puncture.

Thirty-second day: Onset of generalized meningitis.

Thirty-fourth day: Death (autopsy specimen shown in figures 4, 5, 6 and 7).

Comment.—The petrositis began on the nineteenth day. The complications of petrositis were as follows: on the twenty-fifth day, pain in the area of the trigeminus nerve; on the twenty-eighth day, paralysis of the abducens nerve, and on the thirty-second day, meningitis.

My next case with the syndrome occurred in February, 1930.

Case 2.—A boy, aged 12, had otitis media on the left side and spontaneous perforation.

Sixth day: Temperature 103 F.; severe aural pain; myringotomy performed (by my associate, Dr. William Hunt), followed by a profuse discharge, relief from pain and a lower temperature.

Tenth day: Excruciating pain in the left temporoparietal region; temperature 102 F.; profuse discharge; no clinical signs of mastoiditis.

Thirteenth day: Paralysis of the abducens nerve on the left side; excruciating pain in the temporoparietal region; acute illness and signs of meningeal irritation. A mastoidectomy was performed with a wide exposure of the zygoma, and the cells were exposed as far as the superior semicircular canal in order to establish drainage through the antrum-epitympanic route. The cells showed the typical virulent streptococcus engorgement with no destruction of bone. A generous myringotomy was performed in order to maintain drainage through the hypotympanic route.

Fifteenth day: Generalized meningitis and death. No autopsy was permitted, but permission was given to take roentgenograms. Dr. Geyman, the roentgenologist, followed the recent technic of Beer [12] to expose the petrous tip, and he demonstrated pneumatization on both sides; the cells on the affected side were hazy in comparison with the right.

Comment.—The striking symptom was the onset of excruciating pain in the temporoparietal region on the tenth day; here the petrositis began.

I shall cite another personal case to show that the petrositis is not a fanciful involvement, and that when it is overlooked it may lead to serious consequences. This case, which occurred in June, 1923, showed no involvement of the abducens nerve, but it had, I now believe, the other cardinal symptoms of a petrositis, which were so marked that

12. Beer, A. Low: Fortschr. a. d. Geb. d. Röentgenstrahlen **40**:979, 1929.

an abscess of the brain was suspected. The essentials of the history and observations are as follows:

Case 3.—In a boy, aged 17, a myringotomy was performed for acute otitis media on the right side; temperature was 103 F.

Fifth day: Temperature still high; profuse discharge; onset of paralysis in the right side of the face; clinical signs of mastoiditis; mastoidectomy revealed large pneumatic mastoid with engorged cells; hemolytic streptococcus isolated.

Eighth day: Facial paralysis had disappeared.

Seventeenth day: Good postoperative recovery; temperature normal; aural discharge scanty.

Twentieth day: Onset of dull pain in the temporal region; temperature 100 F.; increased aural discharge.

Twenty-fifth day: Persistence of dull pain involving the frontal, temporal and parietal regions; moderate aural discharge; mastoid wound, showing slight drainage, was kept open; temperature between 99 and 100 F.

Thirty-second day: Pain more severe, extending to occiput; patient restless, with poor appetite and temperature of 101 F.; mastoid wound opened widely and found in good condition; myringotomy incision enlarged, followed by profuse discharge.

Thirty-fifth day: Pain persistent, but general condition better; temperature from 99 to 100 F.

Fortieth day: Excruciating pain over entire right side of head; temperature 101 F.; nausea, vertigo and first degree nystagmus. In consultation a question arose as to the possibility of a subdural or brain abscess. An exploration was made, but no abscess was found.

Forty-third day: Death followed generalized meningitis. At autopsy no subdural or brain abscess was found. Autopsy showed a markedly cellular petrous with a large broken-down cell beneath the depression for the gasserian ganglion (fig. 21).

Comment.—The petrositis began on the twentieth day. Instead of the delayed desperate search for an abscess of the brain, I now believe that there might have been some hope in an early exploration of the petrous tip through the antrum-epitympanic route.

Gradenigo, in 1907, collected fifty-seven cases and classified them as follows: group 1, twenty-four with the syndrome; group 2, twenty-nine with the syndrome but complicated by other lesions, such as facial paralysis, optic neuritis, etc.; group 3, four with the syndrome, meningitis and death. I do not believe that it is necessary to consider this syndrome with reference to special groups. In his group there are complications that may arise with any severe infection of the middle ear or the mastoid. I believe that it is better to bear in mind that petrositis may develop from an infection of the middle ear, that this may or may not result in the syndrome, and that intracranial complications may result from a petrositis with or without the syndrome. Furthermore, the syndrome may be associated with other complications, such as a labyrinthitis. That does not mean, however, that the labyrinthitis is the cause of the syndrome; it is merely another of the complications

within the petrous bone. Statistics, however, are interesting, and it is a consoling observation that the majority of the patients recover. Gradenigo's report shows a mortality of only 7 per cent; subsequent reports show the rate to be closer to 18 per cent (Sears [13]).

The important features in the group of patients who have recovered are:

1. The syndrome may appear with no manifestations of mastoiditis, or before, during or after mastoiditis.

2. The course varies: In a few patients only a myringotomy was necessary; the majority were relieved by a simple mastoidectomy. The cases that developed after the operation were more alarming.

3. The pain is usually described as temporoparietal, though it may involve all the branches of the trigeminus. It varies in severity from a mere numbness to a constant excruciating pain. It usually precedes the paralysis and is the first to disappear, usually immediately after the operation.

4. The abducens nerve may manifest only paresis, but usually complete paralysis is present. Recovery is gradual, though some authors have reported complete recovery in a few days after the operation; in other cases the period varied from one to five months.

My first case, which I reported in 1924,[14] is typical of a great number. The severe pain in the frontal and parietal regions disappeared immediately after the mastoidectomy, and the external rectus had normal function within a week.

CONCLUSIONS

1. Pneumatic cells are present in the petrous bone more frequently than is generally known.

2. Two routes of cells extending to the petrous tip are described: (*a*) the antrum-epitympanic and (*b*) the hypotympanic. Because anatomically the cells can always be traced to one of these two routes, embryologically the cells develop in this manner, and clinically the infection may extend from the hypotympanic space as well as from the antrum-epitympanic space. (One must concede the possibility of an infection entering either route near the orifice of the eustachian tube before, or even without, entering the tympanum.)

3. The medical term petrositis is suggested because it centers one's attention on this possible involvement.

4. A petrositis may be present with no obvious clinical signs. One sign suggestive of petrositis is the presence, and especially the recurrence, of a profuse aural discharge in the absence of a definite involve-

13. Sears, W. H.: Gradenigo Syndrome, Laryngoscope **37**:32 (Jan.) 1927.

14. Profant, H. J.: Laryngoscope **34**:140 (Feb.) 1924.

ment of the mastoid, or during postoperative care after mastoidectomy. The first definite sign to appear is pain in the areas supplied by the trigeminal nerve, most commonly in the temporoparietal region.

5. The complications of a petrositis depend on the type and degree of the pneumatization, the virulence of the infection and the resistance of the patient.

6. In the majority of cases Gradenigo's syndrome is a complication of petrositis. (In the other cases, it is a complication of some other complication of the middle ear or involvement of the mastoid, such as a sinus thrombosis, extradural abscess, abscess of the brain, etc.)

7. One must bear in mind that some patients recover after a myringotomy and the majority after a simple mastoidectomy with special attention to the cells about the antrum and zygoma. The radical procedure of exploring the petrous tip is rarely indicated.

CASE REPORTS FROM THE LITERATURE

The following are résumés of case reports published since 1924, with an interpretation of each based on the foregoing study.

CASE 1 (Roberts [11]).—In a boy, aged 16, myringotomy for acute suppurative otitis media was performed on Feb. 15.

Seventeenth day: March 4, clinical signs of mastoiditis; discharge moderately profuse.

Nineteenth day: Temperature 103 F.; white blood cell count 35,500. A simple mastoidectomy was performed; *Streptococcus hemolyticus* isolated; entire mastoid rather large.

Twenty-third day: Temperature normal; for next three days condition satisfactory; freedom from pain.

Twenty-sixth day: March 13, pain in area of fifth nerve (left temple and about left eye); temperature 102 F.; profuse discharge from tympanum and mastoid wounds. The pain persisted, and the temperature ranged between 100 and 102 F.

Thirty-third day: Paresis of sixth nerve.

Fortieth day: March 27, paralysis of sixth nerve; onset of septic temperature: 99 F. in morning and 105 F. in afternoon.

Forty-first day: Temperature normal in morning and 104 F. in afternoon; no meningeal signs; operation on lateral sinus and jugular vein; no clot found.

Forty-ninth day: April 5, temperature gradually became normal.

Seventy-second day: Until this time a slight temperature was present. Paralysis persisted and pain changed to a generalized headache; aphasia.

Seventy-fifth day: May 1, exploration for abscess of the brain gave negative results, and death occurred. Autopsy disclosed that a destructive osteitis on the superior border of the petrous bone near the apex had produced a concavity three-eighths of an inch (0.9 cm.) in depth; a large encapsulated abscess was found in the temporosphenoidal lobe.

Comment.—The petrositis began on the twenty-sixth day, causing acute exacerbation and pain in the area of the trigeminus. Dorello's

canal became involved on the fortieth day. (I believe that the septic temperature which began on this day, and which was present only three times, was due to a bacteremia through the inferior petrosal sinus.)

CASE 2 (Oppikofer [9]).—A woman, aged 48, had an apparently mild suppurative otitis on the right side.

Forty-second day: Pain in the area of the fifth nerve. "Violent headaches that lasted for hours at a time: in the right temple, the occipital and especially the parietal regions. General health remained good; normal temperature."

Forty-fourth day: Mastoidectomy; markedly pneumatized mastoid but only a slight involvement found.

Sixty-eighth day: Whispered voice heard at 2.5 meters. Patient had been sent home, but still complained of violent headaches at times; temperature normal; no intracranial observations.

Eighty-sixth day: Exploration of middle and posterior cranial fossae gave negative results, as did lumbar puncture.

Hundredth day: Temperature 102.5 F.; pulse rate 132; generalized meningitis.

Hundred and fourth day: Death.

Autopsy revealed an extradural abscess on the anterior surface of the petrous bone.

Microscopic sections showed: "Marked pneumatization about labyrinth to the median circumference of internal auditory canal; but only a few small pneumatic cells in the tip; inflammation of mucous membrane with some exudation in lumen of cells; roof of internal auditory canal (beneath extra dural abscess) eroded."

Comment.—A petrositis was complicated by an extradural abscess.

CASE 3 (Russi [15]).—A girl, aged 6, had acute suppurative otitis media on the left side, with spontaneous perforation.

Sixth day: Retro-auricular swelling; also pain in area of fifth nerve: in the temporoparietal region and then over the entire face.

Sixteenth day: Paresis of the sixth nerve.

Twentieth day: A myringotomy was performed. A smear showed staphylococci, streptococci and diplococci.

Twenty-first day: Lumbar puncture gave negative results.

Twenty-second day: Mastoidectomy disclosed purulent granulation in the antrum. The sigmoid sinus was exposed in a small area where there were granulations; the cerebral dura was not exposed. A considerable portion of the posterosuperior wall of the canal was removed in order to obtain better drainage.

Twenty-fourth day: Temperature normal; pain almost gone.

Twenty-seventh day: Sixth nerve normal.

Comment.—There was early involvement of the petrous bone; drainage was obtained by the antrum-epitympanic route.

CASE 4 (Whitham [16]).—*Syndrome Before Operation:* A woman, aged 23, had bilateral otitis. A myringotomy was performed about February 18.

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15. Russi, Pasquale: Le nevralgie facciali nella "Sindrome di Gradenigo," Arch. ital. di otol. **38**:395 (July) 1927.

16. Whitham, J. D.: Mastoiditis with Gradenigo Syndrome before Operation; Mastoiditis with Gradenigo Syndrome following Operation, Laryngoscope **37**:368 (May) 1927.

Fourteenth day: Myringotomy performed again on the right side; left ear normal.

Twenty-eighth day: Pain in area of fifth nerve; "severe pain in right temporal and frontal region."

Forty-second day: First examination: temperature normal; profuse discharge; no mastoid tenderness; ocular movements normal.

Forty-fourth day: Paralysis of the sixth nerve; severe pain.

Forty-sixth day: April 5, a mastoidectomy was performed. The entire mastoid and petrous bone were extremely cellular, the spaces extending far forward into the zygoma and deeply into the petrous below and mesial to the horizontal canal. There was no exposure of the sinus or dura.

Fifty-first day: Pain and paralysis had disappeared.

Comment.—The early symptoms showed more involvement in the petrous bone than in the mastoid. A pneumatic petrous bone is suggested.

Case 5 (Whitham [16]).—*Syndrome Following Operation:* In a man, aged 34, with otitis on the right side, myringotomy was performed about May 18. *Pneumococcus mucosus* was found.

Twenty-eighth day: Pain and tenderness in the mastoid region.

Thirty-fourth day: June 22, first examination: temperature 101.9 F.; pulse rate 84; mastoiditis.

Thirty-fifth day: Mastoidectomy performed; tip and zygoma carefully cleaned out.

Forty-ninth day: Patient left hospital; dry canal; posterior wound clean.

Sixty-ninth day: Return of pain in right ear; abundant discharge.

Seventy-first day: Pain in the area of fifth nerve; severe pain over right eye.

Seventy-second day: Paralysis of sixth nerve; pain increased in severity and extending to forehead and temporal region; dry canal; mastoid wound reddened but no pus could be expressed; caloric reactions normal.

Seventy-third day: July 30, secondary mastoidectomy was performed. "Considerable necrosis was found in the region of the antrum. The zygoma and the region of the posterior semicircular canal were cleaned out as far as possible. The dura was freely exposed in the middle fossa and the opening extended as far forward as possible on the anterior surface of the petrous portion of the temporal bone by removal of squamous plate above the zygoma. Probing in the direction of the gasserian ganglion revealed no pus. The impression was gained that the entire petrous bone was pneumatic."

Eightieth day: Pain in area of fifth nerve had disappeared.

Hundred and thirtieth day: Sept. 25, gradual recovery from paralysis of sixth nerve.

Comment.—A secondary operation was necessary because of the petrositis.

Case 6 (Martin [17]).—A youth, aged 18, had suppurative otitis media on the left side.

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17. Martin, G. E.: Acute Suppurative Otitis Media (Left); The Gradenigo Syndrome; Acute Lepto-Meningitis; Death, J. Laryng. & Otol. **41**:525 (Aug.) 1926.

Tenth day: A mastoidectomy was performed. "The mastoid process was very cellular and full of pus, with cells extending forward along the zygoma; a very large extradural abscess was opened, but the dura was not bulging." The hemolytic streptococcus was found.

Seventeenth day: Wound healed; meatus dry; whispered voice heard at 5 feet (1.5 meters).

Twenty-fourth day: Pain in area of fifth nerve, most marked above the eye; whispered voice heard at 18 feet (5.5 meters).

Forty-second day: Paresis of sixth nerve.

Forty-fourth day: Meningitis had developed. Lumbar puncture yielded cloudy fluid, but no organisms were found. Lumbar puncture performed twice daily.

Fifty-first day: Exploration for abscess of the brain gave negative results.

Fifty-sixth day: Death; no autopsy.

Comment.—A petrositis is indicated by the involvement of the fifth and sixth nerves, in spite of the good recovery from the mastoid infection. The meningitis was a complication of the petrositis.

Case 7 (Archer [18]).—A woman, aged 38, had acute otitis media on the right side; myringotomy was performed, and the patient recovered in a few days.

Eighth day: Recurrence of otitis media; myringotomy performed.

Fifteenth day: Pain persisted and spread to temple; scanty aural discharge; temperature 100 F.; another generous myringotomy performed, with no relief.

Sixteenth day: Mastoidectomy was performed. "The antrum was extremely large and deeply located, as were most of the cells from the tip to the zygoma. No pus was found outside of the antrum." Apparent good postoperative recovery.

Twenty-third day: Pain in area of fifth nerve; severe pain in right eye and temple; temperature 102 F. Mastoid wound in good condition. This condition gradually improved.

Thirty-seventh day: Pain in area of fifth nerve; temperature 100 F.; improvement.

Fifty-first day: Severe pain in area of fifth nerve.

Fifty-second day: Paralysis of sixth nerve; temperature 101 F.; mastoid almost healed; ear canal dry.

Fifty-ninth day: Gradual improvement of all symptoms except paralysis of sixth nerve.

Seventy-third day: Gradual improvement of paralysis of sixth nerve.

Complete recovery occurred four months later.

Comment.—The acute exacerbation did not occur in the mastoid (which was healing satisfactorily) but in the petrous bone, as shown by the pain in the area of the fifth nerve and then the paralysis of the sixth nerve.

Case 8 (Greenfield [19]).—A girl, aged 6, had suppurative otitis media on the right side, with profuse discharge.

18. Archer, Palmer: Gradenigo Syndrome, Texas State J. Med. **23**:666 (Feb.) 1928, case 2.

19. Greenfield, Samuel D.: Gradenigo's Syndrome, Arch. Otolaryng. **9**:171 (Feb.) 1929.

Twenty-fourth day: March 12, mastoidectomy was performed. "The zygoma was completely broken down; the structure extended far forward, beyond the auditory canal into the zygomatic root. The mastoid body was extremely large and unusually pneumatic."

Thirty-second day: Patient sent home because of good progress. In a few days, elevation in temperature began to occur; general condition not as good.

Forty-sixth day: April 2, pain in area of fifth nerve; most severe in temporoparietal region; mastoid wound almost healed; profuse discharge from middle ear; temperature 102 F.

Fifty-seventh day: There was paralysis of the sixth nerve. The temperature had persisted and the pain remained severe. Greenfield advised against lumbar puncture because it was apt to disturb the walling off of the localized meningitis.

Sixty-third day: Gradual improvement; pain and temperature less.

Seventy-fourth day: Paralysis of sixth nerve had disappeared.

Comment.—A profuse aural discharge continued, especially during the involvement of the fifth and sixth nerves. A pneumatic petrous bone with a hypotympanic route is suggested.

Case 9 (Phelps [20]).—A boy, aged 6, had bilateral otitis for which a myringotomy was performed. The right ear was normal in five days.

Fourteenth day: Mastoiditis on the left side observed clinically; myringotomy performed.

Eighteenth day: Severe pain in area of fifth nerve: "in and around the left eye." Child cried for hours at a time.

Nineteenth day: Mastoidectomy performed; *Streptococcus mucosus* found; cells all necrotic.

Twenty-third day: Paralysis of sixth nerve; pain still severe and spread over entire face; condition of middle ear improved; mastoid healing well; temperature between 99 and 100 F.

The pain gradually stopped in three weeks; the paralysis decreased in three months.

Comment.—In a case of otitis media, mastoiditis developed on the fourteenth day, petrositis on the eighteenth day and the complete syndrome on the twenty-third day.

Case 10 (Brown [21]).—A man, aged 24, had suppurative otitis media on the left side.

Fourteenth day: Severe attacks of headache.

Twenty-eighth day: May 10, first examination: profuse aural discharge; no clinical evidence of mastoiditis; temperature 99.8 F.

Thirty-fourth day: Paralysis of the sixth nerve; no definite signs of mastoiditis.

Forty-second day: Mastoidectomy performed; mastoid of the sclerosed type; antrum of normal size; contained no pus but only thickened lining (this shows that profuse discharge did not come from the mastoid cavity but most likely from large sublabyrinthine cells).

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20. Phelps, Kenneth A.: A Case of Gradenigo's Syndrome Complicating Mastoiditis, Journal-Lancet **44**:165 (March 15) 1924.

21. Brown, L. Graham: Gradenigo's Syndrome; Mastoidectomy followed by Bilateral Cavernous Sinus Thrombosis, J. Laryng. & Otol. **44**:601 (Sept.) 1929.

Fifty-sixth day: Recovery from paralysis; wound closing normally; no meatal discharge; general condition good; temperature 99 F.

Fifty-seventh day: Severe pain behind left eye; temperature 100.6 F.

Fifty-eighth day: Excruciating pain; projectile vomiting; a definite increase of aural discharge; temperature 103.6 F.; mastoid cavity reopened; further search made for foci of infection, but none found; negative results from lumbar puncture.

Fifty-ninth day: Definite signs of thrombosis of cavernous sinus.

Seventieth day: Death followed septicemia. Autopsy showed thrombosis; "left temporal bone (by mistake) not removed."

Comment.—Early petrositis was first complicated by pain in the area of the trigeminus, and then by paralysis of the abducens nerve; these subsided and were followed later by the third complication, thrombosis of the cavernous sinus.

CASE 11 (Hamilton [22]).—A girl, aged 19, had suppurative otitis media on the left side.

Feb. 15, 1927: Mastoidectomy performed. A cellular mastoid filled with pus was found.

March 9: Ear dry; wound practically healed; patient sent home.

Twenty-fifth day: Fever and vomiting; two days later, a profuse otorrhea but no fever.

Thirty-fifth day: Temperature 101.4 F.; pain in area of fifth nerve; severe pain over the whole left side of face and head; free otorrhea, with an apparently healthy wound.

Thirty-ninth day: Paralysis of the sixth nerve. Second operation: a few unhealthy zygomatic cells found and removed.

Forty-sixth day: Recovery from paralysis of fifth and sixth nerves; no rise in temperature.

Comment.—Hamilton stated: "The involvement of the fifth and sixth nerves would appear to be connected with a failure to remove some diseased zygomatic cells at the first operation." This is the channel for the antrum-epitympanic route.

CASE 12 (Forbes [23]).—A boy, aged 9, had a "severe cold which became progressively worse."

Tenth day: Patient unconscious; temperature 105 F.

Twentieth day: Gradual improvement.

Twenty-fifth day: Pain in area of fifth nerve; "severe pain over left side of head and face;" paralysis of sixth nerve.

Hundredth day.—First examination by Dr. Forbes: no otitis media; no mastoid pain; loss of tactile and thermal sensation on left side of face; mastoiditis on left side shown by roentgenogram; mastoidectomy performed; "the middle ear was sealed off from the mastoid by granulation tissue in the aditus;" long chain streptococcus found.

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22. Hamilton, E. S. Burt: A Case of Gradenigo's Syndrome, J. Laryng. & Otol. **43**:43 (Jan.) 1928.

23. Forbes, S. B.: Gradenigo's Syndrome, Arch. Otolaryng. **7**:363 (April) 1928.

Hundred and twenty-first day: Anesthesia of fifth nerve had disappeared.
Hundred and thirty-fifth day: Paralysis of sixth nerve had disappeared.

Comment.—Otitis media may have occurred during the rhinitis and extended to the petrous bone as well as to the mastoid.

CASE 13 (Simpson [24]).—A girl, aged 19, had had suppurative otitis media on the right side for several weeks, with pain in the area of the fifth nerve; there was severe pain in the temple, and paralysis of the sixth nerve was present early.

Forty-second (?) day: First examination: profuse discharge; mastoiditis.

Forty-fourth day: Mastoidectomy performed; pneumatic mastoid revealed; zygomatic cells diseased; thoroughly exenterated.

Hundred and fourth day: A radical mastoidectomy was performed because of slow recovery and poor hearing; pain and paralysis soon disappeared.

Comment.—The radical mastoidectomy resulted in better drainage of the petrous bone.

CASE 14 (Simpson [24]).—A woman, aged 32, had had suppurative otitis media on the left side for four months; when involvement of the fifth and sixth nerves began was not known. Mastoidectomy; uneventful recovery. Paralysis of the sixth nerve disappeared in three weeks.

CASE 15 (Simpson [24]).—A girl, aged 8, had suppurative otitis media on the right side.

Twenty-first (?) day: Pain in area of fifth nerve, on entire right side of face; paralysis of sixth nerve.

Twenty-eighth day: First examination; mastoidectomy performed; pneumatic mastoid found.

Forty-first day: Good recovery; patient left hospital; profuse purulent discharge from canal still present.

Forty-second day: Temperature 103 F.; patient vomited.

Forty-third day: Neck slightly stiff; spinal fluid cloudy, but no organisms observed. "After lumbar puncture all meningeal symptoms disappeared." Temperature practically normal for several days.

Fifty-seventh day: Reappearance of symptoms of meningitis; hemolytic streptococcus in spinal fluid.

Sixty-fourth day: Death; no autopsy. (Paralysis of the sixth nerve continued throughout.)

Comment.—In this case of pneumatic mastoid there was good postoperative recovery of the wound, but a profuse purulent discharge continued from the middle ear. It is possible that hypotympanic cells led to the petrous bone and that meningitis was secondary to the petrositis.

CASE 16 (Archer [25]).—A man, aged 43, had acute rhinitis and stuffiness in the ears.

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24. Simpson, W. Likely: Gradenigo's Syndrome, Laryngoscope **37**:728 (Oct.) 1927

25. Archer, Palmer: Gradenigo Syndrome, Texas State J. Med. (Feb.) 1928, case 1.

Twenty-eighth day: First examination: Severe pain in left ear, eye and side of face; myringotomy performed for acute suppurative otitis media on left side.

Thirty-first day: Complete paralysis of the left side of the face (seventh nerve); paralysis of sixth nerve; profuse aural discharge but no evidence of mastoiditis.

Thirty-sixth day: Paralysis of sixth nerve diminishing.

Fifty-seventh day: Improvement in condition of all nerves; ear dry.

Hundred and seventeenth day: Complete recovery.

Comment.—The involvement of the fifth, sixth and seventh nerves and the profuse discharge with no mastoiditis may be explained as petrositis occurring most probably by the hypotympanic route.

CASE 17 (Stark [26]).—A man, aged 43, had otitis media on the right side, for which myringotomy was performed about June, 1925; recovery took place in two months. In November, 1926, he was kicked in the right ear by a horse. A week later, rhinitis appeared. In three days otitis developed on the right side, with slight fever; this lasted for three weeks; then facial paralysis occurred. After mastoidectomy the paralysis disappeared. The wound and ear healed in five weeks. Five months later, postauricular swelling reappeared and was incised; this drained for five months.

In November, 1927, the patient was first examined for severe aching pain in the right temporal region (fifth nerve) and paralysis of the sixth nerve which had been present for five days. The temperature was normal and the patient appeared healthy. The right drum membrane was thick and scarred, but there was no discharge from the middle ear or the mastoid scar. Later the same day, the temperature was 103 F.; the spinal fluid was cloudy, with many cells but no organisms.

Second day: Signs of meningitis; 30 cc. of spinal fluid withdrawn; contained many cells but was sterile; marked improvement of condition.

Eighth day: Six spinal punctures had been performed; only a few cells were recovered from the fluid; mild pain and paresis gone.

Tenth day: Ear and mastoid remained dry throughout. Patient left hospital.

Comment.—Stark expressed the belief that the fifth and sixth nerves were involved by extension via the cells to the petrous tip. An interesting feature of this case is the absence of aural or mastoid discharge during the petrositis and serous meningitis. This can be explained by a walling off of the inflammation just as in mastoiditis the aditus at times walls off the middle ear.

CASE 18 (McKinney [27]).—In a boy, aged 7, suppurative otitis media began on the right side about Nov. 14, 1926.

Fourteenth day: Pain in area of fifth nerve: on left side of face to parietal region; paralysis of sixth nerve.

Thirty-third day: Operation performed on tonsils and adenoids.

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26. Stark, W. Berkele: Gradenigo's Syndrome, Minnesota Med. **11**:388 (June) 1928.

27. McKinney, Richmond: Gradenigo's Syndrome, Arch. Otolaryng. **6**:53 (July) 1927.

Forty-seventh day: Mastoidectomy was performed. "All the cells from the root of the zygoma to the tip were necrotic. The diseased area extended backward toward the apex of the petrous portion of the temporal bone, in the region of the semicircular canals."

An uneventful recovery took place. The fifth and sixth nerves became normal within a few days.

Comment.—The patient apparently had a low grade infection; the petrous portion was invaded by the antrum-epitympanic route.

CASE 19 (Dintenfass [28]).—A girl, aged 6, had bilateral otitis media on September 26, for which myringotomy was performed.

Tenth day (October 6): Temperature 102.3 F.; pulse rate 118; general condition poor.

Eighteenth day: Negative roentgenogram obtained; clinical evidence of disease in the bone, which was of extreme pneumatic type. Because of persistent temperature and general debility, bilateral mastoidectomy was performed. Pus was found only in the antrum on each side; this contained staphylococcus and streptococcus.

Twenty-first day: Pain in area of fifth nerve; severe pain in right temporoparietal region.

Twenty-second day: Paralysis of sixth nerve on right side; pain decreased; pulse rate and temperature normal.

Thirtieth day: Gradual recovery of fifth and sixth nerves.

Comment.—The extensive pneumatization suggests pneumatization of the petrous bone.

ABSTRACT OF DISCUSSION

DR. A. C. FURSTENBERG, Ann Arbor, Mich.: Dr. Profant's comprehensive discussion of Gradenigo's syndrome includes an interesting study of the pathologic changes occurring in the temporal bone secondary to suppuration of the middle ear. The term petrositis adequately describes an inflammatory process that not infrequently complicates an acute suppuration of the tympanic cavity. His studies of the temporal bone confirm the observations frequently made at autopsy, namely, the presence of an osteomyelitis throughout the entire petrous process in those skulls in which extensive pneumatization of the petrous process has occurred.

Inflammation near the apex of the petrous process of the temporal bone seems the only logical explanation for the occurrence of Gradenigo's triad. The character of the pathologic change in this region, however, is still a moot question. The majority of these patients recover, so that opportunity for pathologic study of the abducens and trigeminus nerves is usually lacking.

It seems unlikely that violent pain in the region of the trigeminus and paralysis of the abducens nerve are frequently associated with an osteomyelitic perforation of the petrous bone with direct involvement of the nerves from a suppurative process contiguous to them. An extradural abscess or even a localized meningitis at this point would probably seldom resolve spontaneously. Likewise it would be exceedingly difficult to explain the infrequent occurrence of infection of the cavernous sinus if one were dealing with a purulent process immediately adjacent to

28. Dintenfass, Henry: Gradenigo's Syndrome in Mastoid Disease, Arch. Otolaryng. **3**:349 (April) 1926.

the fifth and sixth nerves. The usual surgical technic employed in these cases, the complete mastoidectomy, does not adequately drain this region, yet recovery following operation is the usual occurrence. More amazing still is the large number of spontaneous recoveries without surgical intervention other than paracentesis of the tympanic membrane.

We must not lose sight of the fact that paralysis of the abducens nerve is occasionally observed in aural suppuration, meningitis, abscess of the brain and lateral sinus thrombosis, and that in many instances it cannot be attributable to disease of the apex of the petrous portion. Isolated paralysis of the sixth nerve is of common occurrence in otitis media and cannot be considered a positive indication of involvement of the tip of the pyramid. When, however, it is accompanied by pain in the temporoparietal region, the presence of a petrositis, as suggested by Dr. Profant, is a logical assumption.

It is my opinion that pain in the region of the trigeminus and paralysis of the abducens nerve in acute aural suppurations are the result of edema of the dura mater covering the base of the skull at the points where the nerves pass through it. I hasten to guard this opinion, as it is based on a single observation at autopsy. A patient with a typical Gradenigo's triad died subsequently of a cerebellar abscess. The latter, I believe, was the direct result of a surgical effort to find a suspected abscess in the cerebellum. Postmortem examination revealed an extensive osteomyelitis of the entire petrous process, but no macroscopic or microscopic evidences of meningitis were present. The dura forming the sheath of the nerves at the base of the skull was edematous with round cell infiltration. Virtually an external pachymeningitis was present, although it was a process which probably frequently occurs in adjacent infections of the temporal bone and from which spontaneous resolution is not impossible. It occurs to me that this pathologic change logically accounts for the signs and symptoms referable to the nerves, and that it fits into the principles that govern the course and prognosis of suppuration of the petrous bone with pain in the region of the trigeminus and paralysis of the abducens nerve.

Dr. Emil Amberg, Detroit: Any physician who has had a patient suffering from Gradenigo's syndrome can appreciate the importance of the contribution of Dr. Profant. That a pneumatized petrous bone may facilitate the development of the condition is apparent. The term petrositis is a commendable one. It is a good addition to otologic nomenclature. Dr. Profant has stated that the complete Gradenigo syndrome is relatively rare, and that petrositis may be present with no obvious clinical signs. These remarks open the way to speculation and show how difficult it may become to evaluate the symptoms in their proper light. There seems to be no doubt but that drainage should be established by a thorough opening of the mastoid. When, however, the complete or incomplete Gradenigo syndrome appears after a mastoid operation, the decision whether expectant treatment should be followed or whether surgical intervention should be instituted is difficult. I published a report of such a case in the *Detroit Medical Journal* (May, 1920) in which further surgical treatment proved unnecessary. Recently I learned of another case. In the first of these two patients the paralysis of the abducens nerve had developed before the mastoid operation and had lasted for about three months. The other patient developed paralysis of the abducens nerve and oculomotor and other disturbances after the mastoid operation. I heard that the paralysis of the abducens nerve cleared up in the course of about eight weeks.

It appears that one is between Scylla and Charybdis. A thorough secondary operation is by no means simple and may even be dangerous. I call attention to

the two patients of Jansen who were operated on with success and whose cases were reported in the textbook of Jansen and Kobrak (Practical Otology for Physicians, 1918). Dr. Eagleton's important paper on the "Unlocking of the Petrous Pyramid" (read in Chicago, March 3, 1930), which has not been available to me, and which I know only through its discussion, may throw some light on the question. Of course, it must be considered whether a complete secondary operation that reaches the tip of the petrous bone may not interfere with the nature of a circumscribed meningitis. This, in my opinion, is the main point in question. Proper judgment concerning secondary surgical intervention may mean everything to the patient. Viewed from this angle, any contribution that adds to knowledge of this important condition merits appreciation.

LOCAL ANESTHESIA FOR THE RADICAL MAXILLARY OPERATION

AN IMPROVED TECHNIC OF ADMINISTRATION *

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AND

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The radical maxillary operation has been performed under local anesthesia by a number of operators, but the technic of administering the anesthetic is by no means standardized, varying as it does with each individual clinic, so that a great many methods have been employed. Some of these have been selected with the idea of infiltrating the tissues at the local site of the operative field; others with the idea of utilizing a combination of the block and infiltration methods. Most of these methods are unsatisfactory, because they distort the anatomic relationship of the cheek to the canine fossa, and especially because the innervation of the structures operated on is such that anesthesia by purely local infiltrative methods fails to produce complete relief from pain at distant foci. For example, during the process of removing bone with the gouge, while no pain may result at the site of manipulation, a great deal of pain occurs in and about the hard palate and teeth.

If satisfactory anesthesia is to be produced, all sensation must be removed from the buccal mucous membrane, the outer and inner walls of the antrum, the upper teeth, the hard palate, the soft tissues overlying the antrum and the lateral nasal wall. By considering the nerve supply of these structures and developing a technic whereby the nerve trunk to each of these tissues is narcotized by local nerve block anesthesia, we succeeded in utilizing a method which results in the complete anesthesia of the areas just named. This method has been used in 125 cases with excellent results, even the most apprehensive patients rarely complaining of pain, and permitting the operation to be carried through to its termination without a single interruption for further application of the anesthetic solution.

METHOD

A preliminary hypodermic injection of $\frac{1}{100}$ grain (0.6 mg.) of scopolamine and $\frac{1}{4}$ or $\frac{1}{6}$ grain (16 or 11 mg.) of morphine is given two hours before the operation is begun.

* Submitted for publication, Sept. 23, 1930.

The technic of our procedure can best be described by considering, as we go along, the nerve supply of the various structures. A 2 per cent solution of procaine hydrochloride containing a small quantity of epinephrine is injected at each site, with a Cook dental syringe and needle and Cook carpule.

STEP 1.—*Injection into the Infra-Orbital Nerve.*—The prominence of the apex of the canine tooth is taken as a landmark, and the needle is inserted in the mucobuccal fold, lateral to and above the point between the first and second bicuspids. It is carried upward and slightly laterward for a distance of about 1 inch in order to reach the infra-orbital foramen, at or near which point the periosteum is penetrated. The injection of a few drops of the solution anesthetizes the following structures: the anterior wall of the maxillary sinus supplied by the anterior superior alveolar branch of the maxillary, which is given off from the infra-orbital at its exit from the infra-orbital foramen. This nerve innervates, in addition, the nerves of the incisor and canine teeth as well as the anterior part of the lateral wall of the inferior meatus and the floor of the nasal cavity; it

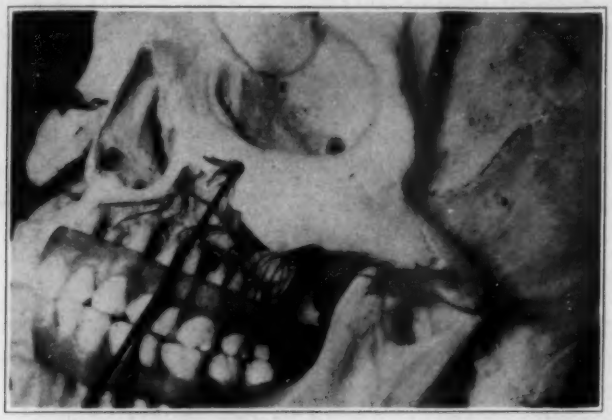

Fig. 1.—Place of injection of the infra-orbital nerve.

communicates, too, with the middle superior alveolar branch. In addition, the injection of fluid at this point anesthetizes the buccal mucous membrane about the upper jaw. An alternative method for the injection, which on occasion we have found useful, is to place the point of the needle in the mucobuccal fold between the two central incisors, carrying it upward and lateralward at an angle of 45 degrees to the level of the infra-orbital foramen.

STEP 2.—*Injection into the Posterior Superior Alveolar Branch of the Maxillary.*—The lips are retracted laterally, the second molar tooth is taken as a guide, and the needle is inserted in the mucobuccal fold and carried upward, inward and backward for about 1 inch. At this point the periosteum is penetrated, and a small quantity (from 0.25 to 0.166 cc.) of the solution is injected. This is termed the "tuberosity injection." At this site the nerve under consideration consists of one or two trunks. It arises from the maxillary at the infra-orbital fissure just before the main trunk enters the infra-orbital groove. It descends on the tuberosity of the maxilla, supplying the mucous membrane of the gum of the upper jaw and cheek and entering the posterior alveolar canal. It runs anteriorly in the bone, giving off branches to the greater part of the lining membrane of the maxillary antrum and a twig to each molar tooth. It is obvious, therefore, that

injection at this site anesthetizes, in part at least, the molar teeth, the gums, the cheek and the lining of the antrum. It is this step, in combination with the preceding one, that permits the painless retraction of the soft tissues, which is not true of the purely local infiltrative methods.

STEP 3.—*Injection into the Anterior Incisive Canal.*—At a point immediately back of the space between the two central incisors, injection is made into the

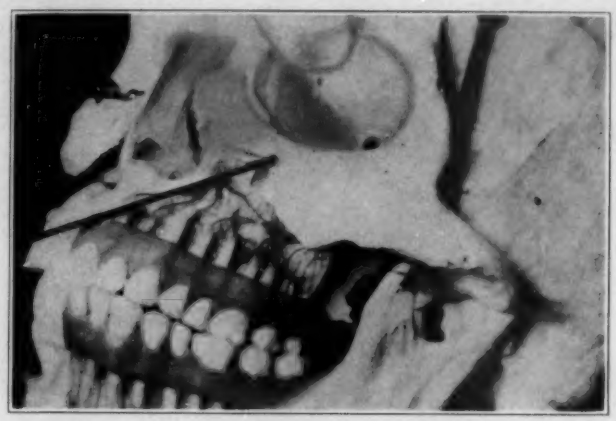

Fig. 2.—An alternative site for the injection of the infra-orbital nerve.

Fig. 3.—Place of injection of the posterior superior alveolar branch of the maxillary.

anterior incisive canal by carrying the needle upward and backward for about one quarter of an inch. Through this canal the nasopalatine nerve passes from the sphenopalatine ganglion to be distributed to the roof of the mouth. Injection at this site relieves pain that would otherwise be experienced over the hard palate during the removal of bone and the curetting of the lining membrane of the floor of the antrum.

STEP 4.—*Injection into the Greater or Posterior Palatine Foramen.*—The needle is inserted superiorly in a depression in the mucous membrane just medial to the third molar tooth, thus reaching the greater palatine foramen, and injection of a few drops of procaine hydrochloride is made. Through this foramen passes the anterior or palatine nerve, descending from the sphenopalatine ganglion and reaching the foramen by way of the pterygoid canal. This nerve supplies in part

Fig. 4.—Place of injection of the anterior incisive canal.

Fig. 5.—Place of injection of the posterior palatine foramen.

the lateral nasal wall, reaching this point through openings in the palatine bone. These latter branches are separate from the main trunk and are called the posterior inferior nasal nerves. They supply the greater part of the inferior turbinate, which, although distant, is therefore partially anesthetized by injection at this site. The main trunk of the anterior palatine distributes itself on the hard palate so that blocking of this trunk, together with the step immediately preceding, removes practically all sensation from the entire hard palate during the chiseling of bony structures and curettement of the lining membrane of the antrum.

As an added precaution and before beginning anesthetization within the nose, a few drops of the solution are injected immediately under the mucous membrane at the frenum. The purpose of this is to relieve pain from traction exerted toward the opposite side with the retractor and because of the free anastomosis of the nerves across the midline.

STEP 5.—*Injection into the Middle Meatus.*—The next point of attack is anesthetizing the lateral nasal wall. For our purpose the nerve supply of this structure can be considered to be derived from the ethmoidal branches of the nasociliary nerve, the anterior alveolar branch of the maxillary and the anterior palatine which comes from the sphenopalatine ganglion. The posterior superior nasal branches also have their origin in the sphenopalatine ganglion. Because of the difficulties involved and the possibility of complications to the eyes following the injection of the sphenopalatine ganglion itself, it has been found convenient to block the branches derived from it by local infiltration of a few drops of the solution into the soft tissues of the lateral wall of the middle meatus. The branches

Fig. 6.—Place of injection of the middle meatus.

which do not arise from the sphenopalatine ganglion are, of course, anesthetized at this site, although the injection of their main trunks has been carried out in earlier steps; in this way the inferior turbinate becomes anesthetized without specific attention to it.

We have operated on patients in whom the middle meatus was the seat of much polypoid degeneration and in whom the middle turbinate had been removed. This made injection difficult and prevented the proper dissemination of procaine hydrochloride throughout the area. At operation, the patient complained of slight pain in the orbit while curetting the roof of the mouth and medial wall of the antrum, but usually this was not sufficiently severe to necessitate further anesthetizing measures, except in rare cases in which topical applications of cocaine and epinephrine were made inside the antrum. These cases illustrate the great importance of making the injections into the middle meatus properly. It is this procedure which, we believe, gives us results superior to those we have observed in other methods of administering local anesthesia for the radical maxillary operation. In many of the cases described, bilateral Caldwell-Luc operations had been performed. On one side, in which the middle meatus was normal and injections could be carried out properly, there was no pain, the roof and medial wall having

been completely anesthetized. On the other hand, in which polypi, marked congestion or impingement of the middle turbinate against the lateral nasal wall occurred, or previous operations had been performed, injections and dissemination were made difficult, and there was inevitably pain while the roof and the lateral nasal wall were curetted.

STEP 6.—*The Use of a Pack Under the Inferior Turbinate or Inferior Meatus.*—Because of the extensive operative procedure often necessary throughout the entire length and the infarction of the inferior turbinate, this structure is most easily anesthetized by placing a strip of gauze saturated with equal parts of a 10 per cent solution of cocaine and a 1 : 1,000 solution of epinephrine under it along the lateral nasal wall. A solution of procaine hydrochloride injected at this site, instead of the cocaine sponge, can be used in some cases. However, since most of these patients have had previous multiple punctures or window operations, the structures are macerated, and injections are thus impossible. The cocaine sponge is allowed to remain in place until the antronasal wall is about to be removed. The

Fig. 7.—Point under the inferior turbinate or the inferior meatus at which the pack is placed.

inferior meatus is supplied by the anterior superior alveolar branch of the maxillary, the anterior palatine from the sphenopalatine ganglion and smaller twigs from this same structure.

As soon as the inferior meatus is packed, the operation is begun. These combined steps take no longer than two or three minutes. No difficulty is experienced in making injections at the proper sites, provided the topical anatomy is well understood and constantly borne in mind.

COMMENT

It can readily be seen, then, that by carrying out these procedures, not only the actual site of operation is anesthetized but the surrounding structures as well, which is not true when the infiltrative method is used. It is to be noted, particularly, that there is no pain when the soft tissues are retracted, and that the upper jaw and teeth are likewise anesthetized. Manipulations about the inner wall of the antrum are painless, and we

have not found it necessary in any case to make injections into the sphenopalatine ganglion.

The advantages of this method are: 1. The complete cooperation of the patient is maintained. 2. There is a minimum of bleeding. 3. It is rarely necessary to halt the operation in order to anesthetize the interior of the antrum with packs of cocaine and epinephrine, which is bothersome and often difficult to accomplish on account of the bleeding. 4. Postoperative swelling is not more marked than when a general anesthetic is used and much less than when infiltration is used. 5. The entire anesthetization can be accomplished with 2 or 3 cc. of a 2 per cent solution of procaine hydrochloride. 6. There is freedom from pain in the adjacent structures, as well as over the operative site. This is never true when local methods other than nerve block are used. 7. It is not necessary to place gauze packs within the oral cavity to prevent insufflation of blood into the larynx. 8. This method offers a definite, systematic technic based on the neuro-anatomy of the region under discussion.

As a concluding statement, it is to be emphasized that proper injection into the middle meatus and the packing under the inferior turbinate are of paramount importance. Failure to anesthetize either site properly because of technical errors or anatomicopathologic changes results in imperfect anesthesia, especially over the roof of the mouth and the medial wall of the antrum.

We have performed a number of operations on the frontal sinus under a similar combination block infiltration method with equally good results. A communication describing the procedures involved is now in the process of preparation.

1915 Spruce Street.

UNLOCKING OF THE PETROUS PYRAMID FOR LOCALIZED BULBAR (PONTILE) MENINGITIS SECONDARY TO SUPPURATION OF THE PETROUS APEX

REPORT OF FOUR CASES WITH RECOVERY IN THREE *

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The chief objects of this communication are to show that from the character of the temporal bone (1) infection of the marrow-containing petrous apex is a different pathologic process—a true osteomyelitis—from suppuration of the pneumatized cells of the mastoid; (2) that different principles of treatment are consequently applicable, and (3) that in a surgical attack on the apex, the squama and tympanic ring may be removed, as all the external portion of the temporal bone is solely for protection of the neural tissues of the labyrinth and the brain.

SURGICAL PATHOLOGY

The temporal bone, from a physiologic standpoint, is composed of three portions. 1. The first is an external protective portion made up of the squama, tympanic ring and mastoid with its pneumatic cells.

The squama and tympanic ring are membrane bones which phylogenetically are a secondary type of bone, while the mastoid portion is the lateral arm of the primordial chondrocranial base which surrounds the vital neural structures of the bulb, and is developed for their protection.

During the first three years of life the mastoid becomes pneumatized, the pneumatic spaces being lined by nonciliated epithelium, an outgrowth of the epithelium of the eustachian tube and tympanic cavity. There are no true bone-marrow spaces in the mastoid, certainly not after its pneumatization in infancy. The tympanic cavity, which originates in the cartilage of bronchial arches, is closely associated with the respiratory functions.[1]

2. The middle third of the temporal bone contains the functioning portion: the labyrinth, the end-organ of the labyrinth and cochlea.

3. The internal third of the temporal bone contains an intracranial space-developing portion, the apex cells, containing blood-forming mar-

* Submitted for publication, Sept. 2, 1930.

* An extension of the paper presented before the Eastern Section of the American Otological, Rhinological and Laryngological Society, New York City, Jan. 4, 1930, and the Chicago Otolaryngological Society, Chicago, March 3, 1930, by the addition of two later cases.

1. McMurrick, J. P.: The Development of the Human Body, ed. 6, Philadelphia, P. Blakiston's Sons & Company, 1915, p. 449.

row substance morphologically the same as the marrow of the long bones. The petrous belongs to the more primitive type of cartilage bone [2] (chondrocranium).[3]

INFLAMMATION OF THE PNEUMATIC SPACES OF THE MASTOID

Embryologically, the mastoid process appears as the posterior buttress of the periotic capsule to which it becomes united, as the mastoid with the chondrifying mesenchyme of the occipital and sphenoidal regions joins to form the continuous chondrocranium [4] which surround the primitive vital nervous centers.

At birth, the cancellous tissue of the undeveloped mastoid is filled with fat and, like other infantile bones, probably contains marrow substance; but the later growth of the mastoid process is by pneumatization in order to furnish lightness to the compact bony arches of the sides and front of the skull. The mastoid cells thus are (*a*) pneumatic spaces lined by epithelial prolongations from the mucous membrane of the middle ear and the eustachian tube. The cells are separated by (*b*) delicate bony trabecula. The mucous membrane of the eustachian tube, middle ear, antrum and mastoid cells are all anatomically outshoots of the upper respiratory tract. The ciliated glandular type of epithelial lining gradually gives place to a squamous and columnar non-mucous-producing form as it leaves the nasopharynx and passes out of reach of external irritants.

The bony tissue surrounding the mastoid cavities is but supportive and protective.

Wolff's law, that the bony lamellae are laid down according to the stress to which the bone is subjected, applies to the mastoid process up to the time of birth; this takes place in order to protect properly the nervous structures during the passage of the child's head through the pelvis.

A careful examination of the outer protective portion of the temporal bone (mastoid, middle ear and squama) fails to reveal any true blood-forming bone-marrow,[5] although it often persists in the squama.[5a]

In the tympanic cavity the arteries go directly into the veins without the intercession of capillaries;[6] this is conducive to the prevention of

2. Cunningham: Text-Book of Anatomy, ed. 5, New York, William Wood & Company, 1923, p. 292.

3. McMurrick (footnote 1, p. 180).

4. McMurrick (footnote 1, p. 171-172).

5. Hagens, E. W.: Acute Suppuration of the Temporal Bone, Arch. Otolaryng. **10**:21 (July) 1929.

5a. McMahon, B. J.: Personal communication.

6. Prussack: Zur Physiologie und Anatomie des Blutstromes in der Trommelhohle, Berichte der königlich sachsichen Gesellschaft der Wissenschaft, 1868; Arb. a. d. physiol. Anst. zu Leipz. **3**:86, 1869.

infection, but the blood supply of the mastoid comes from the submucosa of the mucous membrane and from the vasa vasorum of the adjacent tissues. Consequently, in infection of the mucous membrane of the cavity of the middle ear, suppuration rarely goes on to bony destruction, while in infection of the mastoid bony destruction is the rule.

If infected, the anatomic construction of the mastoid is favorable to the development of a diffuse carious process followed by cavity formation filled with pus and granulation, the extent of the cavity being prescribed by the limiting compact osseous tissue or by the adjacent mesothelial venous sinus wall or dura.

MASTOIDITIS AND OSTEOMYELITIS

The pathologic sequence in mastoiditis inflammation is swelling and round cell infiltration of the epithelial lining followed by caries with pus and granulation formation, the granulation springing from the diseased bone itself, of which it forms a part.

Kopetsky's [7] coalescent mastoiditis is a suppurative osteitis of a pneumatized bone—it is not a true osteomyelitis, as there is no true bone-marrow [8] in the mastoid process.[9]

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7. Kopetsky, Samuel J.: Systemic Infection Complicating Purulent Middle Ear Disease, J. A. M. A. **83**:336 (Aug. 2) 1924.

8. Myelitis, from the Greek variant of muelon, variant of muelos-marrow, Concise Oxford Dictionary, p. 536.

9. Josef Fischer (Studien zur pathologischen Anatomie des Schlafebeines, Monatschr. f. Ohren., 1925-1926, vols. 59 and 60), like most authors, regards osteomyelitis as an inflammation of the substance of the bone. As long as the bony, which he regards as haversian, and Volkmann's canals are attacked, he speaks of osteomyelitis without any regard to whether "bone marrow" is normally present in the canals or not. He also disregards the fact that although there are bony spaces in the petrous and mastoid, which resemble the haversian canals of the long bones, the peculiar method of development of the periotic capsule as well as the unique pneumonic character of the mastoid process make it questionable whether these spaces are true haversian canals. All his sections of suppurative lesions (figure 35, "acute purulent periostiles"; figure 37, "acute osteomyelitis"; figure 39, "acute osteomyelitis with necrosis"; figure 40, "chronic periostitis ossificans"; figure 41 and figure 42, "chronic osteomyelitis"; figure 43, "chronic osteomyelitis with necrosis"; figure 44, "chronic osteomyeltis"; figures 45 and 46, "chronic osteomyelitis with healing by bone formation") are sections through the middle ear or the labyrinth. The condition or presence of bone-marrow is not mentioned. As none of these regions normally contain marrow, certainly not after pneumatization, none of his cases can be regarded as really cases of osteomyelitis.

Only figure 38 ("acute osteomyelitis from scarlet fever otitis"), in which the section shows a subperiosteal abscess in the base of the middle fossa, can possibly qualify as inflammation in a region containing medullary substance.

Proper classification is of more than academic importance as disease in or of the marrow cells of a bone is different from that of the cancellous and compact structures of a pneumatic bone.

On the other hand, bone-marrow occurs in the sphenoid, a cartilaginous bone, the spongy part of which does not become pneumatized until the eighth year. Consequently, either the roof of the air sinus or the body of the sphenoid itself may be the seat of osteomyelitis with the characteristics of an infection of the bone-marrow itself. This has been demonstrated microscopically by Turner and Reynolds.[10]

There may be spontaneous recovery from coalescent mastoiditis so long as there is no disorganization of the bony tissue with pus formation. Clinically, the suppurative course in coalescent mastoiditis is that of a low grade infection, while involvement of the sinus or leptomeninges is a later process from pressure necrosis. This occurs largely because the inflammation is usually caused by infection from nonhemolytic organisms.

When, however, the micro-organism is of the hemolytic type, the infection is apt to attack simultaneously the small blood vessels, the venous plexuses of the sinus or of the leptomeninx. In "hemorrhagic mastoid," the suppuration extends by a retrograde thrombophlebitis or perivasculitis, and early intracranial complications are frequent.

UNIQUE CHARACTER OF THE BONE OF THE PERIOTIC CAPSULE

The bony labyrinthine otic capsule, with which the apex develops, is different from other cartilaginous bone in that it forms the only complete capsular bone of the human skeleton, and it also has the unique property that it contains islands of cartilage-like cells that do not become ossified throughout life.[11]

The apex resembles the metaphysis of a long bone in structure and function, and consequently in its pathologic condition resembles the metaphyseal end of a long bone. Anatomically, instead of a central nutrient artery, its blood supply is from the periosteum loops, the nutrient arteries of the apex being twigs sent from the carotid plexus to the compact cortex,[12] similar to the blood supply to the cortex of a long

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10. Turner, A. Logan, and Reynolds, F. Esmond: A Study of the Paths of Infection to the Brain, Meninges, and Venous Blood Sinuses from Neighboring Peripheral Foci of Inflammation, J. Laryng. & Otol. **43**:568 (Aug.) 1928, fig. 3.

11. Bast, T. H.: Osteogenesis of the Human Periotic Capsule, Arch. Otolaryng. **10**:459 (Nov.) 1929.

12. Koerner, C.: Applied Anatomy of the Ear, in Denker, A., and Kahler, O.: Handbuch der Hals- Nasen- Ohren- Heilkunde, Berlin, Julius Springer, 1926, p. 204.

bone from the periosteum.[13] The apex is the active growing part, the intracranial space-developing portion of the temporal bone. The apex continues to possess a peculiarity, for while at birth the size of the bony labyrinth is that of an adult,[14] the petrous apex continues to grow until late in life, long after the epiphysis of the long bones have been firmly united to their diaphysis, as demonstrated by the ease with which the petrous apex can be disarticulated (unlocked) from the base of the skull even late in life.

ENLARGEMENT OF THE BASE OF THE SKULL BY GROWTH OF THE PETROUS APEX

The petrous apex grows horizontally toward the median line of the skull and laterally parallel with the base of the skull. It is the growth of the petrous apex and of the occipitosphenoidal base (the original chondrocranium) that widens and deepens the cranial fossa. The growth of the apex not only expands the base, but largely dictates whether or not the labyrinthine capsule shall have pneumatic cells on its anterior and upper surface (peritubal region), its posterior (superior petrosal area) or its lower (mastoid tip-digastric-jugular region) surface, because the pneumatic cells which surround the bony labyrinth are formed solely to fill in the spaces left by the lateral expansion of the apex.

The continued increase in the size of the cranial cavity is made possible by a membranous layer which at birth separates the cartilagum petrous from the osseocartilage of the occipital behind and of the sphenoidal base in front. Until advanced life, the mesial surface of the apex and a considual part of its anterior and posterior borders are surrounded by cartilage. It is this anatomic peculiarity, this continuation of the activity of the growing end of a modified long cartilaginous bone (Poland[15]), that is the factor that makes the apex liable up to ten years to the type of osteomyelitis[16] (Siebenmann) that is only encountered in the long bones during early childhood.

But the most important element in the transmission of infection is that the petrous apex is the only region of the body in which pneumatized cells containing noncitrated epithelium may be normally

13. Karig: Acute Suppurative Osteomyelitis, Thesis, Graduate School, Surgical Section, University of Pennsylvania, 1926.

14. Tremble, G. B.: Bony Labyrinth of the New-Born Infant and of the Adult, Arch. Otolaryng. **9**: 175 (Feb.) 1929.

15. Poland, John: Traumatic Separation of the Epiphysis, London, Smith, Elder & Company, 1898.

16. Siebenmann, F.: Otogenic Osteomyelitis in Children Taking a Fulminating Course, Cor.- Bl. f. schweiz. Aerzte **49**:1737, 1919.

brought, by pneumatization, into contact with the medullary substance of a blood-producing bone without a layer of bone and a mucous-producing protective mechanism intervening. For it is the mucous-producing ciliated epithelium which lines the pneumatic spaces of the accessory nasal sinuses that protects the bone of the nasal sinuses from infection. These three anatomicophysiologic peculiarities, (*a*) periosteal blood supply, (*b*) growing bone and (*c*) exposure of marrow-containing bone substance to direct infection, not only influence the cause and character of any suppurative lesion that may attack the apex, but also, I believe, render the apex especially liable to infection reactions in mastoiditis, as well as providing it with unusual facilities for its control. For I am persuaded that Gradenigo's syndrome—facial pain and abductor paralysis in the presence of a suppurative otitis—is caused by a combination of an infective congestive lesion of the apex cells and a protective reaction in the meninges. In the vast majority of the cases, the bone spontaneously controls the infection because of its highly reactive character.

OSTEOMYELITIS OF THE PETROUS APEX

The petrous apex is not a pneumatic, but a marrow-containing, bone, similar to the active growing end of the long bones of the skeleton; its cells are small spaces filled with medullary substance.[17]

Embryologically, the petrous apex is a part of the bony otitic capsule; it becomes ossified with the labyrinthine capsule from the "prootic" (Huxley) osseous center.[18]

Normally, the matrix[19] of the apex is filled with cellular marrow placed between the layers of compact bone which form the carotid canal in front, the floor of the middle fossa above and the posterior surface of the petrous behind. Exceptionally, pneumatic cells fill the apex[20] in which case the pneumatization process is engrafted onto the medullary substance by a direct extension from the middle ear either through the peritubal region or along the posterior surface of the pyramid, in a similar manner that the membranous squama or the cartilaginous occipital may become pneumatized.

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17. See illustrations of petrous apex, figure 1, case 1.

18. Gray, Henry: Osteology. Temporal Bone, in Anatomy, Descriptive and Surgical, ed. 20, London, Longmanns, Green & Company, 1918, p. 146.

19. Brooks, B., in discussion on Bancroft, F. W.: Fate of Necrosed Bone in Chronic Osteomyelitis, Tr. Sect. Surg., General & Abdominal, A. M. A., 1922, p. 154.

20. Mangabeira-Albernaz, P.: Large Pneumatic Cell of the Petrous Bone: Contribution to the Pathogenesis of the Gradenigo Symptom-Complex, Arch. internat. de laryng. **8**:1035, 1929. This author discovered a specimen in which the apex was one cell, 29 mm. in length, 14 mm. in height, and 9 mm. in width.

PATHOLOGY OF OSTEOMYELITIS OF CAPSULAR AND FLAT CARTILAGE BONES OF THE SKULL IN CONTRAST TO OSTEOMYELITIS OF THE LONG (PIPE) BONES

Osteomyelitis of the long bones of children is a clinical entity and different from that of adults. Acute hematogenous osteomyelitis in children is a metastatic, "artero-thrombo-embolic phenomena" [21] which occurs in growing bone in certain areas (generally the metaphysis of the most actively growing end).

The infective process is usually controlled by simple incision, because it is in growing bone and is thus surrounded by the active reparative properties of the medullary and periosteal surfaces.

Chronic infective osteomyelitis of a long bone of an adult is always the result of a direct infection into the bone, such as from compound fractures. It is much more difficult to control because of the absence of the active growing properties of the child's bone.

Acute osteomyelitis of the flat bones, such as occurs in osteomyelitis of the frontal bone following operations on the frontal sinus, follows direct extension of suppuration in the bone. It travels in the diploic tissue and causes nutritional death. It is apt to recur, often remaining quiescent for long periods and then advancing again. It is usually limited by the sutures.

Chronic osteomyelitis of small and flat bones is generally caused from a chronic nutritional disturbance of the bone following syphilis, tuberculosis, actinomycosis or in phosphorous poisoning and diabetes.

CHARACTERISTICS OF OSTEOMYELITIS OF THE APEX

In the petrous apex there is a combination of growing periosteal-nourished bone as pertains in the active end of the long bones of infants, and liability to infection from suppuration of adjacent tissue as occurs in compound fractures by the veins or by extension through the pneumatic spaces surrounding the labyrinthine capsule. The result is a peculiar type of osseous suppuration in which the protective reaction modifies the destructive lesion. For the type of the osteomyelitis is dictated by the characteristics of the apex, viz., that of a growing end (metaphysis), the nutrient supply of which comes from multiple small vessels entering from the periosteum; to this, micro-organisms are brought directly from suppuration in the region of the tympanic mastoid. The infection, whether it is from a throm-

21. Lexer, E.: Die Enstehung entzündlicher Knockenherde, Arch. f. klin. Chir. **71**:1, 1903. Wilensky, Abraham O.: The Mechanism and Pathogenesis of Acute Osteomyelitis, Ann. Surg. **82**:781, 1925; Am. J. Surg. **3**:281, 1927. Eagleton, Wells P.: Infective Arthritis Complicating Otitis Media (case 4), Arch. Otol. **34**: 483, 1905.

bophlebitis of small veins or suppuration within perilabyrinthine cells, causes local nutritional death of the tissue with a marked surrounding protective reaction from the marrow and growing bony tissues. The amount of pus formation is generally small, but a cavity may be formed filled with a granulation mass with areas of pus, the mass resembling a tumor formation. The latter occurs because the inflammation is in marrow-filled cancellous tissue, which is actively growing. A similar condition occurs from overstimulation of osteoclasts by trauma or other disturbing factors while intracartilaginous bone is being formed with the production of a giant cell tumor, instead of normal new bone.

Recent studies on bone pathology suggest that bone cysts of the metaphysis, which occur in the early decades of life, are the result of a healed process of growing cartilage bone, and that giant cell tumors of the epiphysis are due to hyperactivity of the osteoclasts from an irritant.[22] There is probably a definite chain of abnormal bony changes, varying from chronic hemorrhagic osteomyelitis to Barries' hemorrhagic tumor, to fibrous osteomyelitis,[23] ostitis fibrosa, bone cysts and giant cell tumors [22] of the medulla of cartilage bone. And there is some evidence that bone cysts are of bactericidal origin.[24]

GRANULATIONS IN MASTOID DISEASE AND IN OSTEOMYELITIS OF THE PETROUS APEX

The differences between the two pathologic processes of caries of the mastoid and osteomyelitis of the apex is seen in the character of the granulation tissues formed.

In mastoid disease, the granulations are nature's effort to limit the infection; they spring from, and are a part of, the bone, and to remove them the layer of the bone from which the granulation comes must be forcibly curetted.

In osteomyelitis of the apex, the granulating mass is the reaction of the surrounding infected growing bone in an effort to fill a cavity caused by nutritional death.

The granulations of osteomyelitis of the medullary substance of a long bone are called by the French, "fongosities"; they are "overgrown

22. Geschеckter, C. F.; Copeland, M. M., and Bloodgood, J. C.: Ostitis Fibrosa and Giant Cell Tumor, Arch. Surg. **19**:169 (Aug.) 1929.

23. Phemister, D. B.: Chronic Fibrous Osteomyelitis, Ann. Surg. **90**:756, 1929, abstr., Arch. Path. **9**:917 (April) 1930.

24. Santos, J. V.: Bacteriology of Apparently Normal Bones, J. Bone & Joint Surg. **12**:150, 1930. Phemister, D. B., and Gordon, John E.: The Etiology of Solitary Bone Cyst, J. A. M. A. **82**:1311 (April 26) 1924.

poorly nourished oedematous masses which when cut do not bleed as freely as normal granulations." [25]

In two of my cases of suppuration of the petrous apex, the masses were compressed by the overlying dura and laid almost free in the bony cavity, from which they could be lifted easily. They contained giant cells (Dr. Lewis Brown).

In suppuration of the apex, an involucrum is never formed; this is characteristic of all flat bones.[26] A sequestrum of the apex forms but rarely, but a sequestrum of the osseous labyrinth [27] is far from infrequent, especially in tuberculosis. Such a sequestrum occurs because interior contents of the osseous labyrinth are supplied by one nutrient artery, although the bone itself receives some blood from the small vessels of the carotid plexus.

TREATMENT SUGGESTED BY THE PATHOLOGY OF OSTEOMYELITIS OF THE APEX

It is the combination of a destructive infective lesion in the medulla of a rapidly reproductive bone which in chronic suppuration of the apex offers a favorable field for the control of infection by simple drainage of the infecting area much as infantile osteomyelitis of the long bones is cured by drainage and bone cysts are cured by incision and giant cells by curetting, the elimination by removal of all areas of the disease, such as is demanded by suppuration in the mastoid, being unnecessary.

CLASSIFICATION OF OSTEOMYELITIS OF THE TEMPORAL BONE

My experience and a review of the literature on osteomyelitis lead me to suggest the following classification for osteomyelitis of the petrous apex, based on recognized types of osteomyelitis of the long and flat bones.

Acute Hemorrhagic Osteomyelitis of the Temporal Bone of Infancy and Childhood in Growing Bone.—This type of osteomyelitis is rare; I have encountered it but once. It occurs only in infancy and childhood, for at birth the cancellous spaces of all the bones are filled with fat. However, it may occur at a much later age (at least up to the tenth year) in the temporal bone than does the acute hemorrhagic osteomyelitis of the long bones, because of the longer period of cartilaginous

25. Spurrier, E.: Joint Sequestra of Osteomyelitis, J. Bone & Joint Surg. **11**: 747, 1929.

26. Kahlke, in Ochsner, J. A.: Surgical Diagnosis and Treatment, Philadelphia, Lea & Febiger, 1920, vol. 1, p. 316.

27. Eagleton, Wells P.: Vestibular Tests in Intracranial Surgery (case 6), Tr. Am. Laryng. Rhin. & Otol. Soc., 1922.

bony expansion of the apex. It occurs chiefly in girls from 1 to 7 (Siebenmann), while acute hemorrhagic osteomyelitis of the long bones occurs much more frequently in boys.[28]

Acute hemorrhagic osteomyelitis may involve all parts of the temporal bone, the squama and the mastoid, as well as the apex. The disease is an acute infective arteothrombo-embolic phenomenon, as is shown by the fact that it jumps over areas of bone, leaving them healthy.[29]

The mortality rate is very high, and the condition rapidly results in sequestration, especially of the bony labyrinth.[30] Acute hemorrhagic osteomyelitis of the temporal bone is comparable to the acute hemorrhagic osteomyelitis of the metaphysis of the long bones of infants.

Chronic Osteomyelitis Suppurative and Reparative of the Petrous Apex.—This type of osteomyelitis is due to direct extension of an infection to the (1) periosteum of the petrous pyramid or (2) into the medullary substance.

The infection reaches the apex by way of (*a*) a retrograde thrombophlebitis of the perilabyrinthine veins, (*b*) through the labyrinth or (*c*) by way of the pneumatic cells which surround the labyrinth.

Clinically and pathologically, the disease may be chiefly limited to a periostitis of the apex or may be a medullary infection of the apex.

1. Periostitis of the Apex: If the infection involves only the periosteum, as occurs in many of the peritubal cases (case 4), there will result a superficial erosion of the superior surface of the petrous, on the floor of the middle fossa. Here a local extradural abscess may form, which may be microscopic in size. The extradural abscess is generally situated under the gasserian ganglion or over the carotid artery [31] because of the venous anastomosis to the venous carotid plexus and cavernous sinus.

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28. Ochsner, A. J., and Crile, D. W.: Osteomyeltis, Surg. Gynec. Obst. **31**: 263, 1920. "In 104 cases in the Copenhagen Hospital, there were three times as many boys as girls."

29. Turner and Reynolds (footnote 10). Figure 3 shows a large area of acute purulent necrosing osteomyelitis of the sphenoid roof, adjacent to which is normal bone-marrow, which latter "shows no signs of reactions." Siebenmann (footnote 16).

30. Georg Jung reported a case of sequestrums of the whole labyrinth and apex on both sides in an infant. Doppelseitige symmetrische Einschmelzung des Schläfenbeines, des Warzenfortsatzes und fast der ganzen Felsenbeinpyramide, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:267, 1928.

31. Eagleton, Wells P.: Cavernous Sinus Thrombophlebitis, New York, The Macmillan Company, 1926, case F, p. 88. A case is reported of thrombophlebitis of the jugular vein and bulb of the left side, from the left tonsil, associated with right-sided otitis media; necrosis of squama, mastoiditis, labyrinthitis and double purulent sphenoiditis. Postmortem examination revealed diffuse osteomyelitis of the spheno-occipital bases and "an area of perilabyrinthine cells filled with pus, situated at the apex of the petrous pyramid just behind the carotid artery.

2. Medullary Infection of the Apex: When the medullary cells are attacked by the infection, (*a*) a congestion results, following which the extent of the normal reactive processes of the bone and the virulence of the micro-organisms will dictate whether the inflammation causes macroscopic or microscopic (*b*) caries or (*c*) abscess. The destructive lesions, however, are modified by the reparative process of the actively growing bone.

3. Granulation Tissue Formation: The reparative process is evinced by granulation tissue formation, the granulation being of the type of a giant cell tumor compressed by the surrounding intact dura. Therefore in the majority of the cases in the literature at postmortem examination the cortex was but slightly discolored and the medulla softened.

4. Medullary Osseous Healing in the Cartilage Bone of the Periotic Capsule: The reparative process in infective lesions of the periotic capsule occurs in the interior of the bone, not by a hyperactivity of the periosteum, but through obliteration of the cavities by new connective tissue followed by new bone production. This is because of the specific type of the bony structure and its growing properties. The tendency to new bone formation is more marked in the phylogenetically later cochlear than in the semicircular canals and vestibule.[32]

CLINICAL TYPES OF OSTEOMYELITIS OF THE APEX

(*a*) *Retropharyngeal Abscess of Petrous Origin.*—The under surface of the apex is rough and the compact tissue deficient in places. This deficiency accounts for a few of the cases of retropharyngeal abscess from otitis in infants, in whom the marrow cells of the apex are well developed at birth, long before the pneumatization of the mastoid process, for "growing bones develop abscesses of the type of osteomyelitis within their metaphysis. Adult bones do so but rarely." [33]

In the usual lymphatic retropharyngeal abscess in infants as a complication of otitis, recovery occurs after drainage of the abscess and proper treatment for the otitis. Recovery occurs also when the retropharyngeal suppuration is the result of osteomyelitis of the apex, there being no extensive bony destruction because there is little confinement of the infection; the lack of compact tissue allows immediate discharge of the pus into the soft tissues. Osteomyelitis of the petrous apex with retropharyngeal abscess is in reality a Brodie's abscess. The disease is directly below the periosteum and does not involve the medulla.

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32. Kasae, T.: Experimental Studies on Meningogenic Labyrinthitis Curative Processes, Japan M. World **6**:144, 1926.

33. Robinson, quoted by Beekman: Ann. Surg. **82**:227, 1928; J. Bone & Joint Surg. **9**:8, 1927.

(*b*) *Lateral Pharyngeal Abscess from the Apex.*—After infancy, the location of the suppuration in the bony area at the base of the skull formed by the rough petrous apex and the attachment of the levator veli palati to its anterior part determines the situation in the upper lateral pharyngeal wall that a dissecting abscess from the apex uniformly assumes, as well as its flat shape.

I have twice encountered such a lateral pharyngeal abscess in adults. A case in which there was a fistulous tract below the labyrinth connecting it with the middle ear was exhibited before the New York Otological Society in March, 1930, by Dr. Bowers. Roentgenograms showed the flat anteroposterior character of the abscess.

In adult life, lateral naso-pharyngeal abscess from suppuration of the petrous apex runs a slow course. In one of my cases all the mastoid cells were filled with "plasma" cells such as are frequent in giant cell tumors. The plasma cells apparently grew outward into the pneumatic spaces of the mastoid and later covered the sinus, giving the clinical appearance of a neoplasm.[34]

(*c*) *Intermedullary Infection of the Petrous Apex.*—The inner surface of the apex articulates with the basal process of the sphenooccipital bone, to which it becomes completely united very late in life. This explains why confined suppuration originating in the apex is sharply limited to the petrous, never extending to the adjacent basilar plate (fig. 1).

However, osteomyelitis of the occipitosphenoidal bases with sphenoidal suppuration may extend to the apex,[35] the result of a retrograde thrombophlebitis which extends outward from the cavernous sinus to the veins draining the apex.

Macroscopically, osteomyelitis of the petrous apex frequently shows only "congestion."[36] And in two reported autopsies, the opposite pyramid gave a similar appearance, although there were no symptoms or contralateral otitis to account for it.[37]

However, in the fatal cases that are "simply discolored" the microscope reveals round cell infiltration (case 1) and even abscess formation

34. Phemister, D. B.: Unusual Forms of Osteomyelitis, Northwest Med. **27**: 460, 1928. "In the skull there are slow localized destructive types of osteomyelitis in which fibroblasts, giant cells and special types of leucocytes predominate. There may be eosinophiles. This form of osteomyelitis has been mistaken for tumors, as myelonias."

35. Eagleton (footnote 31). Turner and Reynolds (footnote 10). Jacques and Lucien: Bull. et mém. Soc. franç. d'oto-laryng., March, 1908, p. 146. A case of osteomyelitis of the sella of the sphenoid and apex of the petrous from an acute cavernous sinus thrombophlebitis, the result of a peritonsillar abscess, is reported.

36. Heine: Berl. klin. Wchnschr. **27**:769, 1900. von Trölish, A. F.: Arch f. Ohrenh. **4**:121, 1868.

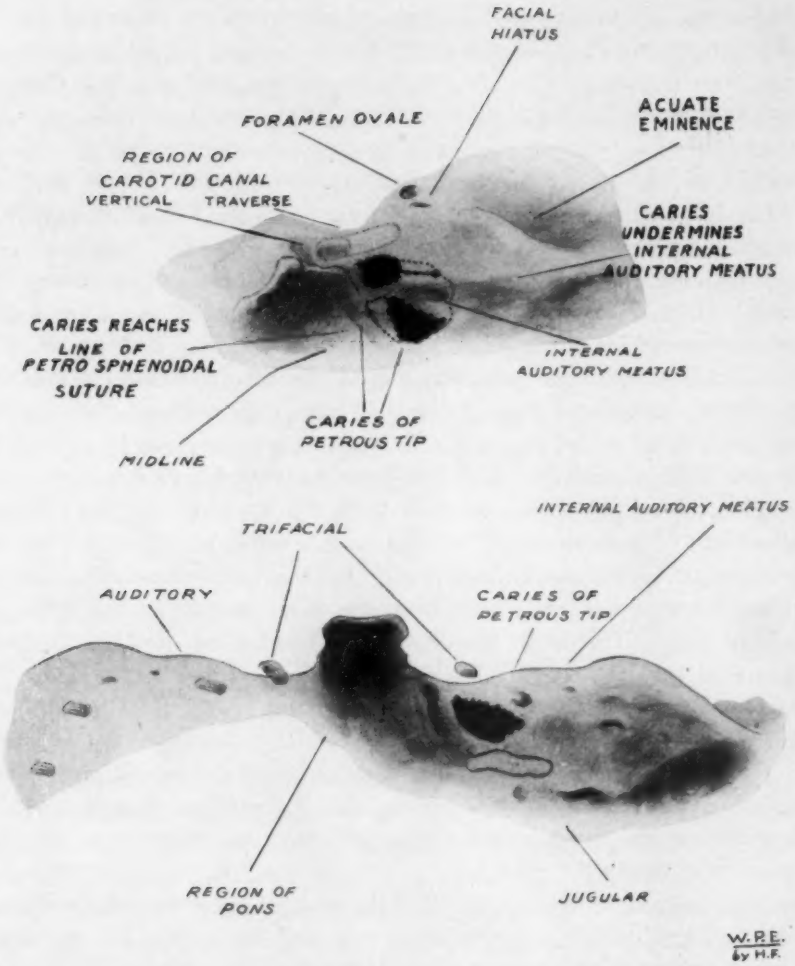

Fig. 1.—Drawing showing the extent and limits of the necrosis and cavity formation in a case of suppuration of the petrous apex (case 2, A. R., former paper), which is fairly typical of several of the recorded cases. The necrosis perforates the cortex in both the middle and posterior fossae, a bridge of compact bone of the superior border being left between them (Lombard and Baldenweck, L.: Étude anatomique et clinique sur relations de l'oreille moyenne avec la point du rocher le ganglion de Gasser et la sixième paire cranienne, Paris, G. Steinheil, 1908, case 24, pp. 114-115. Knapp, Arnold: Primary Thrombosis Cavernous Sinus, Arch. Otol. **35**:437, 1906.) The cavity undermines the internal auditory meatus but is sharply limited by the petrobasal articulation. The upper drawing is of the superior surface; the lower one, of the posterior surface. In both of my cases there was no perforation of the dura which macroscopically was intact. The tense dura compressed the granulations so that they gave the appearance of a "new growth" filling the cavity in the apex. (Berget and Piqué: Osteite tuberculeuse du rocher, Bull. Soc. anat. et gaz. méd. de Paris, July, 1885, no. 28, p. 328.)

of the medullary substance.[38] It is noteworthy, however, that like all cases of chronic, so-called fibrous osteomyelitis, the infection may remain confined to the medulla of the bone for long periods of time, death eventually occurring from general leptomeningitis, the perforation of the compact tissue of the cortex being microscopic in size.[39]

In those cases in which the inflammation of the marrow of the petrous apex is of long standing, granulation tissue, pus formation and necrosis of the compact bone result. The perforation occurs either on the (*a*) upper surface (into the middle fossa under the gasserian ganglion), at which point a local extradural abscess exceptionally develops or (*b*) through the posterior compact layer to the dura of the posterior fossa. When the posterior surface of the bone is attacked, the suppurative process involves either the bulbar pontile cisterna or the cisterna of the angle.

Here the suppuration may continue for some time before it causes a terminal general leptomeningitis, although that portion of the bony cortex of the apex which forms both the middle and posterior fossae may be extensively eroded, the apex cavity being filled with firm granulations tightly confined by dura, as in cases 1 and 2 (McE. and A. R.).[40]

In contradistinction to suppurative mastoiditis, extradural abscess formation is relatively rare,[41] although perforations of the cortex occurs.[42]

37. Noltenius: Rev. heb. de laryng., 1905, no. 48. In this case the bone of the right petrous tip (the side of the affected ear) "was not normal in color. The dura was so adherent to it that it tore when detached." The disease of the apex was secondary to a bony necrosis of the cells of the roof of the eustachian tube, where there was a small area (size of a pea) of superficial brain necrosis. The opposite (L) petrous tip at about the same place was similarly "changed in color with evident lesions less marked than on the right side, although during life the patient had never complained of his left ear."

38. Schlander: Ztschr. f. Hals-, Nasen- u. Ohrenh. **12**:492, 1925.

39. See figure 1 and case 2, A. R. Case 1 lasted for seven weeks.

40. Eagleton, Wells P.: Localized Bulbar Cisterna (Pontile) Meningitis, Facial Pain and Sixth Nerve Paralysis and Their Relation to Caries of the Petrous Apex (Cases 1 and 2), Arch. Surg. **20**:386 (March) 1930.

41. Sheppard, J. E.: Arch. Otol. **26**:268, 1897. In this case there was an abscess over the foramen lacerum medium. Heine (footnote 35, case 1). Hilgermann, Robert: Die Beteiligung des Ganglion an der Pyramidenspitze, Ztschr. f. Ohrenh. **40**:315, 1901-1902, cases 3 and 6. All cases had abscess formations in or under Meckel's cave. Buckner: Arch. f. Ohrenh., vol. 19, p. 349, case 3. In this case there was an abscess below the fifth nerve and an adhesion of the cerebellum at this point.

42. Picqué, L., and Février, C.: Contribution à l'étude des abcès intra-crâniens d'origine otique; anatomie pathologique; pathogénie; traitement, Ann. d. mal. l'oreille, du larynx **18**:883, 1892, ob. 2. In this case there was an abscess on the posterior surface. Eklund, Thurel: Ueber die Abduzenslähmung als Komplikation der Mittelohreiterung, Finska läk-sällsk. handl. **58**:632, 1916; abstr., Arch. f. Ohrenh-, Nasen- Kehlkopfh. **100**:73, 1916-1917. In this case there was an extradural abscess from peritubal cells.

There may even be a microscopic abscess under the gasserian ganglion in the middle fossa without macroscopic evidences of pus formation.[43]

SURGICAL INDICATIONS IN OSTEOMYELITIS OF THE PETROUS APEX

The surgical indication in osteomyelitis of the petrous apex, at least in the early stages, is simply to enter the medulla of the bone by a sufficiently large opening through the bony cortex, as is done in acute osteomyelitis of the long bones, by incision through the compact outer layer, thus cutting short the process in the medulla itself.

For even when pus is formed it is not necessary to eradicate the whole infected area, as in disease of the pneumatic bone, because the marrow itself has great reparative properties. Likewise, prolonged drainage is unnecessary, as it has been demonstrated that osteomyelitis of the long bones is best treated by "saucer evacuation" and local sterilization at the time of operation and subsequent rest (leave alone) treatment (Orr's treatment [44]). The efficiency of such treatment in osteomyelitis of the apex is illustrated by cases 2 and 3 of this report.

MODE OF INVASION OF INFLAMMATION OF THE PETROUS APEX

Of the six personal cases of suppuration of the petrous apex, including cases 1 and 2 of my previous paper [40] and four cases in the present report, there is clinical evidence that the petrous apex was invaded by way of the blood stream in five, the usual method in osteomyelitis of the long bones. All four patients had one or more chills prior to the appearance of the meningeal symptoms, and in infections

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43. Schlander, E.: Zur Histologie der Eiterungen an der Pyramidenspitze, Ztschr. f. Hals-, Nasen- u. Ohrenh. **12**:492, 1925. Engelhardt, G.: Abducens Paralysis in Extradural Abscess at the Apex of the Petrous Bone, After Acute Middle Ear Suppuration; Death from Meningitis, ibid. **11**:194, 1925. Ulrich, K.: Zur Lehre von der otogenen Abducenslähmung, ibid. **9**:403, 1925. Ulrich reported a case in which the entrance of infection to the meninges was demonstrated only on microscopic examination. The postmortem examination showed that the original focus was extradural and affected the tip of the petrous bone. The gasserian ganglion was invaded and materially damaged by suppuration, and the sixth nerve was reduced in size to a mere thread. Karlefors, John: Fall von Meningitis, ausgegangen von Zellen in der Spitze der Pars petros, Acta otolaryng. **4**:372, 1922. In this case, the dura was microscopically free. Microscopically, there was purulent infiltration in the dura around the trigeminus, hyperemia and numerous granulations cell forms; lymphocytes, plasma cells, isolated leukocytes and possibly fibroblasts. The cells of the petrous apex under the dura and of the trigeminal region were filled with granulations.

44. Patterson, M. B., and Albee, Fred: Bacteriophage in Relation to Healing of Osteomyelitis, Proc. Soc. Exper. Biol. & Med. **27**:376, 1930. Orr, H. W.: Osteomyelitis and Compound Fractures, St. Louis, C. V. Mosby & Company, 1929.

in the head, I have come to regard a chill as indicative of a blood stream infection.[45]

In the few cases in the literature in which the petrous apex has been examined microscopically, thrombosis of the veins, especially of those surrounding the carotid, the carotid venous plexus,[46] has been found in several [47] while in others the postmortem observations would warrant such an interpretation.[48]

The infection may possibly have reached the petrous apex by direct extension of the suppurative process through pneumatic cells of the peritubal region in one case (case 1, O. R., former series),[49] as there was operative but no postmortem evidence of infection in the tubal region of the middle ear; however, as the patient suffered from repeated chills, it is fair to infer that the disease of the apex was of blood stream origin. In one case (case 4), the operative observations warrant the belief that the infection reached the apex by way of the veins on the superior surface of the pyramid.[50]

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45. Eagleton, Wells P.: Brain Abscess from the Standpoint of the Otolaryngologist, Laryngoscope **40**:336 (May) 1930.

46. Spalteholz, W.: Handatlas der Anatomie des Menschen, Leipzig, S. Hirzel, 1913, p. 397, B. 442.

47. Schlander, E.: Zur Histologie der Eiterungen an der Pyramiden-Spitze, Ztschr. f. Hals-, Nasen- u. Ohrenh. **12**:492, 1925.

48. Hilgermann, Robert: Ztschr. f. Ohrenh. **40**:311, 1901-1902. Sheppard, J. E.: Arch. Otol. **26**:268, 1897. In this case, at postmortem examination an epidural abscess was found in relation with the foramen lacerum; the bone in the region of the carotid canal was softened, and as the canal was thought to be the root of the infection, the carotid venous plexus probably transmitted the infection. Heine (footnote 35, case L). In this case there was a perforation of the bone in the superior surface of the petrus involving the carotid canal and undermining the posterior surface, and as the carotid artery and the labyrinth were unaffected, the infection probably ascended by the carotid venous plexus. Hilgerman (footnote 41, second reference, case 6). In this case there was purulent thrombosis of the different vessels of the cavernous sinus. Eagleton, Wells P. (footnote 31, case D, p. 72). At operation there were granulations around the internal carotid artery, and at postmortem examination a partial clot was found in the artery.

49. Uffenorde, W.: Zur Klinik der Failo von Mulelober mit teefen perilabyrinthisren Herdig (case 1), Arch. f. Ohrenh. Nasen- u. Kehlkopf. **105**:107, 1918-1920. In this case a report is made on sections of the petrous apex showing suppuration. The infection apparently came from the tubal region; there was secondary involvement of the labyrinth. Noltenius (footnote 36). In this case there was necrosis of the roof of the tube.

50. Pietrantoni, L.: The Routes of Propagation of Inflammatory Processes from the Middle Ear to the Apex of the Petrous Bone, to the Cavernous Sinus, the Sixth Pair and the Gasserian Ganglion: The Venous Route, Arch. ital. di otol. **38**:296, 1927. The author has anatomically demonstrated that infection may reach the petrous apex from the middle ear by venous communications which pass along and with the anterior superior surface of the petrous, as in many bones

In one case (case 3, G. D.), there was definite operative evidence that the suppurative disease extended to the petrous apex from the mastoid region by way of a succession of cells which occasionally pass inward from the mastoid above the jugular bulb but below the entrance of the saccus endolymphaticus and the internal auditory canal.[51]

Labyrinth Infection in Suppuration of the Apex.—In two of my cases the labyrinthine involvement was probably secondary to the infection of the apex and not the cause of it (case 2, McE., former series, and case 1, M. G., this report). In one case (case 3, G. D.), the labyrinth was undoubtedly injured by the operative procedure. Ultimately, there was complete recovery. Consequently, in four of the six cases, the labyrinth was not attacked by the disease (case 1, O. R., former series; case 2, B. L.; case 3, G. D., and case 4, A. W.).

PERIOD OF SEMIQUIESCENCE OF INFECTION IN THE APEX AND OF THE ASSOCIATED LOCALIZED BULBAR (PONTILE) MENINGITIS

As previously pointed out,[40] the terminal general leptomeningitis, from which all persons with suppuration of the petrous apex finally succumb, is frequently preceded by a long period during which there are frequent attacks of meningeal symptoms due to the efforts that nature makes to localize the infection in the bone and to the bulbar (pontile) and angle cisternas.

In one case there were symptoms suggesting infection of the petrous apex for forty-three days (case 1, O. R., previous series); in another, for sixteen days (case 2, McE.), and in a third case, for seven weeks (case 1, M. G.). Of the three cases herein reported, in which there was recovery, one patient was operated on seven weeks after the appearance of meningeal symptoms (case 2, B. L.), another on the sixteenth

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this area is pneumatized and "is always traversed by a fine reticular venous plexus which is externally continuous with the submucous venous plexus of the tympanum and internally with the numerous venous trunks of the apex. Of these, the median veins anastomose with the pericarotid venous plexus, while the anterior become united with the plexus which surrounds the gasserian ganglion and with them open into the cavernous sinus."

51. Hilgermann (footnote 40, third reference). In this case there was a "fine tract" of cells from the hypotympanum through the jugular bulb cells. Lombard and Baldenweck, L.: Étude anatomique et clinique sur relations de l'oreille moyenne avec la pointe du rocher, le ganglion de Gasser et la sixième paire cranienne, Paris, G. Steinhel, 1908. In Lombard's case, the infection occurred "through the fine tract" of pneumatic cells from the antrum along the posterior surface above the internal auditory meatus with a large cavity in the petrous apex. The cavernous sinus was thrombosed. In Baldenweck's case, there was a "fine tract" originating just above the auditus and going along the superior surface of the petrous above the labyrinth which was not involved.

day of the meningeal involvement [52] (case 3, G. D.), and the third one month after her fifth nerve was first irritated (case 4, A. W.).

This confirms my previous contention that, as a rule, osteomyelitis of the petrous apex and its associated localized meningitis give outspoken symptoms for a sufficient time for a localizing diagnosis to be made and surgical treatment to be given.

CLINICAL COURSE OF SUPPURATION OF THE PETROUS APEX WITH AN ASSOCIATED LOCALIZED BULBAR CISTERNA MENINGITIS [39]

All of my cases have run a typical course and have presented a terminal picture that is characteristic.

The patient has an otitis followed by a mastoid operation, from which he does not get entirely well, although for a considerable time he is not very sick. During the postoperative period he has headache which occasionally is severe, especially during the night, at which time it may be associated with the "significant pain behind the eye." [53] During the quiescent period, an external rectus paralysis is frequent. This is generally diagnosed as "Gradenigo's syndrome," for which a mastoidectomy is performed, the operator believing this to be all that is necessary as in 80 per cent of the cases Gradenigo's syndrome signifies a "congestion" of the apex cells plus the protective reaction of the dura which is relieved by a complete exenteration of the mastoid. However, the seriousness of the condition should be suspected, even at an early stage, as, unlike the usual Gradenigo external rectus paralysis, suppuration of the petrous tip is associated with low grade sepsis manifested by a slight and irregular rise in temperature, from 99 to 101 F., with sweating at night, and by attacks of a slightly stiff neck, lumbar puncture revealing either clear or slightly cloudy fluid which is sterile.

In two of my cases, that is, one third, labyrinthitis suddenly developed, the outspoken symptoms of which further obscured the picture (McE., case 2, former report; M. G., case 1, present series).

Suddenly, the temperature rises, the patient becomes delirious, and the fluid contains streptococci. This general streptococcic leptomeningitis is a terminal process and invariably results from a long continued localized meningitis of the bulbar cisterna.

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52. Roberts, E. R.: Acute Otitic Temporosphenoidal Abscess, Septicemia, Gradenigo's Syndrome, Acute Surgical Mastoiditis, Acute Suppurative Otitis Media, Fronto-Ethmoidal Empyema, Grip: Report of a Case with Postmortem Observations, Arch. Otolaryng. **6**:420 (Nov.) 1927. In this case there were symptoms of suppuration of the apex with meningitis for four months before death.

53. Eagleton, Wells P.: Sixth Nerve Involvement in the Diagnosis and Surgical Treatment of Meningitis of Otitic and Nasal Origin, Tr. I., Congrès International d'oto-rhino-laryngologie, Copenhague, 1928, p. 822. Von Tröltsch first described this significant pain behind the eye (footnote 53, second reference). Ostman: Relations of Eye to Ear, Arch. f. Ophth., 1897, vol. 43, no. 1.

SYMPTOM-COMPLEX OF BULBAR (PONTILE) CISTERNA MENINGITIS

During the terminal stage, all of my patients presented a symptom-complex characteristic of pontile cisterna meningitis, viz.: The patient is unconscious but not stuporous. He lies on his back with his head slightly extended and his eyes closed. When spoken to, he arouses and will answer questions and then dozes again. There is some headache but little of the agonizing pain, and none of the general restlessness and screaming of cortical meningitis. In a few days he dies.

Autopsy in all of my cases revealed either a caries or an inflammatory congestion of the petrous apex from which there had developed a localized inflammation of the cisterna over the pons. The original focus of infection in the petrous apex had continued to feed the meningeal inflammation until the latter had extended over the cortex.

With the exception of the cases herein reported, all cases of suppuration of the petrous apex with meningitis have ended fatally, as case 2 is the first instance in which the primary focus in the apex has been successfully opened surgically and the localized meningeal collection simultaneously evacuated. However, in 1893, Bircher [54] reported a case of extradural abscess from suppuration of the petrous apex from which the patient recovered. The patient had in reality a case of Brodie's (subperiosteal) abscess of the petrous apex, similar to the condition in case 4.

Also Brockaert,[55] in 1914, successfully opened Meckel's cave for a localized meningitis with neuralgia of the face and abductor paralysis of otitic origin. However, he did not open the apex.

REPORT OF CASES

CASE 1.[56]—*Infection of the petrous apex with localized meningitis, following otitis media and mastoiditis; later labyrinthitis, probably secondary to the petrous apex disease. Terminal leptomeningitis. Ligation of the common carotid; opening of the petrous apex through the nonfunctioning labyrinth; drainage of the lateral arachnoid cisterna of the posterior fossa of the arachnoid prolongation surrounding the internal auditory. Death from general leptomeningitis, probably hastened by disturbance of the cerebral circulation from the carotid ligation.*

History.—M. G., a young man, seen in consultation with Dr. Henry C. Barkhorn at Beth Israel Hospital, Newark, N. J., had acute otitis media followed by hemorrhagic mastoiditis. Operation was done, followed by a stormy convales-

54. Bircher, H.: Centralbl. f. Chir., 1893, no. 22, abstr. by Ballance and Hobbrouse, Tr. Ninth International Otological Congress, Boston, 1912, p. 314.

55. Brockaert, Jules: Supra-Auricular Operation in Cases of Otitic Sixth Nerve Paralysis Associated with Neuralgia of the Trigeminal Nerve, Bull. Soc. franç. otol., 1914, p. 323.

56. Cases 1 and 2 were first reported before the Eastern Section, American Otological, Rhinological & Laryngological Society, January, 1930.

cence without a great rise in temperature but a great deal of pain in the head. There was early blood stream infection, as shown by a cardiac murmur and sweating at night.

Several weeks after the operation on the mastoid, the patient had a severe pain behind his eye. He described it as if "the eye was being shoved out." This later disappeared.

He was discharged from the hospital but continued to have headache of such severity as to necessitate the use of morphine for relief. During all of this period he had a slight sepsis.

Suddenly, a homolateral facial paralysis developed. The wound was reopened, and the cells in the angle between the external and superior semicircular canal were found to be diseased.

Two days after the second operation, the patient presented symptoms of an acute homolateral labyrinthitis; he vomited, had vertigo, nystagmus and became totally deaf on the affected side. There was no paralysis of the sixth nerve. Two lumbar punctures revealed clear fluid.

The vomiting, nystagmus and vertigo disappeared, but he continued to complain, irregularly, of pain in his head which was not severe and was relieved by acetylsalicylic acid, although previously morphine had to be administered.

Seven weeks after the onset of the labyrinthine symptoms, following an unusually happy day (in the evening of which he had played cards), he was suddenly awakened by an agonizing headache. Shortly afterward he became slightly delirious, and his temperature rose to 104 F. Lumbar puncture now showed streptococci in the fluid. The next day the temperature was 106 F., and he had marked symptoms of cerebral intoxication.

A diagnosis of caries of the petrous apex with a secondary labyrinthitis by extension from the tip cells outward and a complicating bulbar cisternal meningitis was made.

Operation (W. P. E. and H. C. B.).—The common carotid was ligated and unlocking of the petrous pyramid was done, with the patient under ether anesthesia, so that the external wall of the labyrinth was fully exposed. Entrance was made into the apex cells by a small electrically driven burr, which was passed through the vestibule and the basal coil of the cochlea, but kept well forward, thus exposing the carotid artery in the transverse portion of its bony canal. There was no loss of cerebral fluid during the intralabyrinthine operation, as the modiolus was not disturbed. This possibly was aided by the presence of a protective exudate in the internal auditory meatus, as there had been a labyrinthitis. Beyond the labyrinth, the burr entered a fair sized cavity filled with granulations and containing a little pus. The depth of the inner wall of the cavity in the petrous apex from the plane of the bony superior semicircular canal was 4 cm. The dura over the posterior fossa was now nicked, and a large quantity of cerebrospinal fluid was drained from the arachnoid prolongation which surrounded the internal auditory meatus.

Subsequent History.—The patient never regained consciousness. He had a continuously high temperature, widely dilated pupils and desquamation of the superficial layers of both cornea from neuropathic keratitis. The blood stream was laden with micro-organisms. He died on the following day of respiratory paralysis.

Autopsy (Dr. Asher Maguda).—Externally, the bone of the petrous apex did not appear necrotic but had a rather grayish appearance. Its interior was carious and contained a drained cavity.

The brain showed general basilar meningitis with edema of the arachnoid of the cortex. There was a purulent exudate along the vessels, especially around the optic chiasm, and a slight amount of exudate over the base of the cerebellum, with pus in the ventricle and covering the choroid plexus. There were multiple small hemorrhages into the substance of the temporosphenoidal lobes, the cerebral tissue appearing softened around the hemorrhagic areas.

There were multiple small thromboses in the veins of the cortex of the hemisphere on the side of the ligation. A large hemorrhage in the piarachnoid on the right side of the brain stem was limited by the basilar artery.

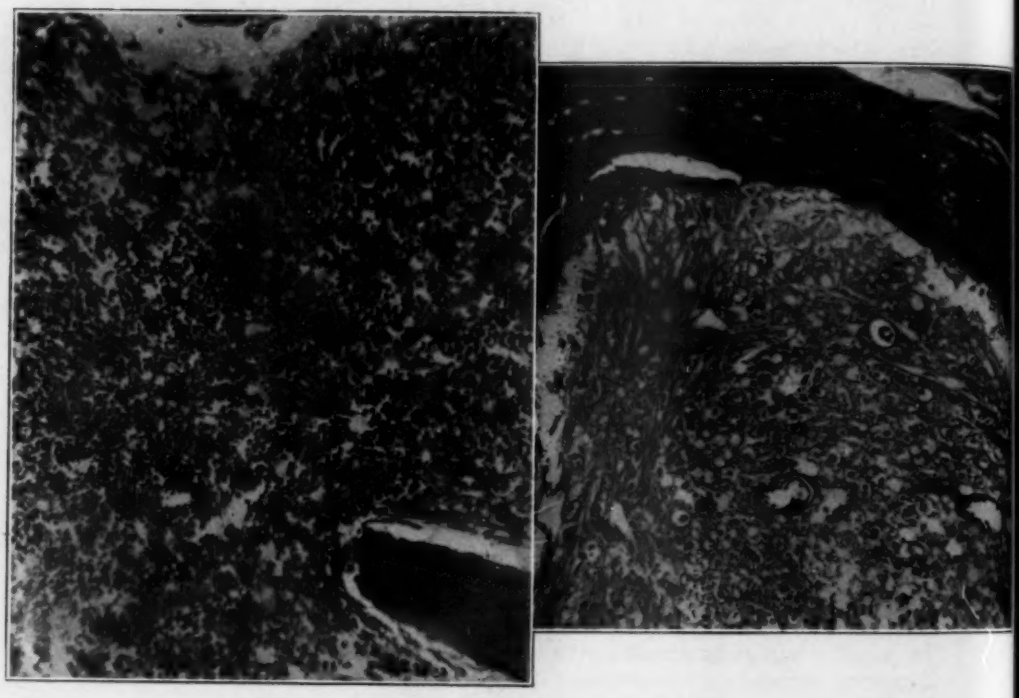

Fig. 2.—Sections of the petrous tips (case 1, J. G., and of case 2, O. R., former series) show "cellular structure in the bone spaces typical of adult red bone-marrow. There is a fine network of blood capillaries in close association with collections of cells. Normoblasts and adult red blood cells are fairly numerous, as are neutrophilic and eosinophilic myelocytes and polymorphonuclear leukocytes. There are typical marrow cells, megakaryocytes, in the sections. These different types of cells are intermingled in close association with each other and with the reticular network of the capillaries." (Legend by Dr. Lewis W. Brown.)

Comment.—Although the patient was in a desperate condition at the time of operation, his failure to regain consciousness, his dilated pupils and the neuropathic keratitis suggested that possibly the ligation of the common carotid had occasioned cerebral edema which had contributed to his demise; therefore, I determined in the future to perform

a preliminary "fractional ligation" (Kerr [57]) under a local anesthesia as on previous occasions.[58]

The distance from the plane of the superior semicircular canal to the apex of the petrous tip, measured by a probe, was 4 cm., and as the examination of a number of adult petrous bones have given a similar measurement, this distance may be adopted as a surgical guide during operation to determine whether or not the end of the petrous apex has been reached.

CASE 2.—*Localized bulbar cisterna meningitis the result of osteomyelitis of the marrow of the petrous apex, from otitis media with mastoiditis; perforation of the petrous apex after passing along the floor of the middle fossa under the gasserian ganglion; evacuation of infected fluid from the posterior fossa; immediate exaggeration of all meningeal signs; prompt recovery.*

History.—B. L., a woman, aged 36, referred to Dr. John V. Bissett by Dr. A. T. Lipphard, on May 14, 1929, had pain in the left ear following tonsillectomy, but examination the next day showed an apparently normal drum membrane. During the first three days of her illness, she had a very severe headache, two chills, some sweating and two attacks of vomiting.

On May 19, the drum was opened, with a slight discharge. The patient improved, but during the next three weeks she continued to have pain in her head, and she had several slight chills.

On June 8, mastoidectomy was performed (Dr. A. L.).

The headache did not disappear, and on the fifth and sixth day after the operation; "the light bothered her eyes," and she was "a little confused." On June 16 (thirty-three days after the initial earache), she complained of double vision from paralysis of the external rectus, felt chilly,perspired and had headache and pain behind the left eye. She vomited several times.

On June 26, a lumbar puncture resulted in clear fluid, with 60 cells, mostly lymphocytes.

On June 28, she had a slightly stiff neck and a slight Kernig sign. Lumbar puncture revealed 128 cells.

On June 29, 1929, she was admitted to the Newark Eye and Ear Infirmary, to the service of Dr. John V. Bissett, her chief complaint being "severe but intermittent pain in the head and double vision,"

Examination.—Examination showed paralysis of the left external rectus, and slight nystagmus on looking in both directions. The results of the ophthalmic examination were negative. She could hear with both ears. Her neck was slightly stiff, and there was a slight Kernig sign. The upper abdominal reflexes were absent. The knee jerks were hyperactive. The temperature was 99 F., the pulse rate, 88.

A diagnosis of meningitis of the bulbar cisterna from caries of the petrous apex was made.

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57. Kerr, H. N.: Fractional Ligation of Common Carotid Artery in Treatment of Pulsating Exophthalmos, Surg. Gynec. Obst. **41**:565 (Nov.) 1925.

58. Eagleton, Wells P.: Localizing Value of Ophthalmic Examinations in Suppurative Diseases of the Brain, J. A. M. A. **92**:713 (March 2) 1929; The Carotid Venous Plexus as the Path of Infection in Thrombophlebitis of the Cavernous Sinus: Its Relation to Retropharyngeal and Sphenoidal Suppuration, Arch. Surg. **15**:275 (Aug.) 1927.

The question was how best to reach the cellular area of the petrous apex, entrance through the labyrinth, as in the former case, being impossible because the labyrinth was functioning.

Operation (J. V. B. and W. P. E.).—Unlocking of the petrous pyramid was performed. The posterior buttress was first removed; the knee and descending portion of the lateral sinus were exposed and freed. The angle of the superior-petrosal was then removed, and the region of semicircular canals exposed. After the sinus was reflected backward, the dura in the niche for the saccus endolymphaticus presented and was incised. The arachnoid prolongation surrounding the internal auditory meatus was opened, and a large amount of cloudy cerebrospinal fluid was evacuated. The latter apparently came from a large cavity between the dura and the anterior and under surface of the cerebellum. Through the opening in the dura, the top of the jugular bulb could be seen. The anterior buttress was now removed, and the dura separated from the middle fossa. The foramen spinosum with the middle meningeal artery was seen, also the third and second branches of the trifacial nerve at their exits from the base of the skull. A large middle meningeal vein in the anterior part of the wound was accidentally torn and profuse hemorrhage followed, which was controlled by packing of the foramen spinosum. The dura of Meckel's cave was elevated, under which the bone of the petrous apex was perforated by a curved hook. The hook entered the bone easily; the compact bone was soft and carious. The apex tip contained a cavity of some size. The soft parts were partially closed, leaving a small rubber strip along the base of the skull for drainage, as well as several small cotton tails that had been placed to control bleeding.

Subsequent History.—On June 30, the patient was in excellent condition, but had a severe pain in the head at intervals and vomited more than is usual from ether. The temperature varied from 99.2 to 102 F.

July 1 to 2: She had all the signs of a general suppurative meningitis. She complained of dizziness and headache and was irrational at times. She had one chill lasting seven minutes; the temperature varied from 101 to 105 F. However, she did not look very sick. Lumbar puncture revealed 3,800 white cells, 9,800 red cells, and a scanty growth of nonhemolytic streptococci on culture. The surgical manipulation had apparently precipitated a general overflow meningitis.

On July 3, there was no pain in the head; the temperature varied from 99.2 to 102.4 F.

On July 4, there was much greater mobility of the right external rectus.

On July 7, diplopia was not present. The patient made a rapid recovery.

CASE 3.—*Localized suppuration of the petrous apex with a localized bulbar cisterna meningitis, the suppuration probably occurring at the same time as the mastoiditis, the infection reaching the apex by a chain of cells which extended along the posterior surface of the petrous, above the jugular bulb. Preliminary ligation of common carotid; unlocking of the petrous pyramid; negative result of exploration of the region of the petrous apex along the floor of the middle fossa; operative discovery of a fine fistula behind the facial canal; evacuation of the suppurating apex cavity by creating a groove in and along the posterior surface of the petrous. Evacuation of cerebrospinal fluid from the bulbar cisterna; secondary serous labyrinthitis from the trauma of the operation; recovery.*

History.—G. D., a girl, aged 12, was seen in consultation with Dr. Walter Tymeson and Dr. George E. Kalter. She had been in bed for one week, suffering from coryza and general aching accompanied by slight temperature. A diagnosis of grip was made.

On February 25, while walking to the bathroom, the child suddenly became dizzy and fainted; shortly afterward she had pain in the left ear. Paracentesis for acute suppurative otitis was performed on the following day.

On March 1, there was a discharge from the ear. There were no signs of mastoid disease, but tenderness along the side of the neck. The child was restless at night because of pain in the ear, neck and head.

On March 4, she complained of violent pain in the left temple; she had a chill, but little fever.

On March 6, she had a second attack of violent pain in the head. The discharge from the ear was more profuse, and the mastoid became tender. At operation (Dr. Walter Tymeson) coalescent mastoiditis with pus was found. The dura was not exposed.

On March 7, there were attacks of pain in the head of the same intense type as had characterized the headache from the beginning; there was also pain in the neck. The temperature was around 101 F.

On March 10, the child had severe pain during the night.

On March 11, her temperature suddenly rose to 105 F., and again descended to 100 to 101 F.

On March 12, she was nauseated.

The next day (one week after operation), there was pain over the second branch of the fifth nerve on the left side. There was but a slight rise in temperature.

On March 14, there was profuse sweating.

On March 15, the child again had an attack of severe pain in the temple and ear. She became drowsy, and had a temperature of 99.3 F.

On March 17, the pain was so agonizing that she had to be given morphine. The pulse rate became slow, 56. She vomited and complained of blurred vision.

Examination (W. P. E.).—The patient held one eye closed as if she had diplopia. She could move both eyes well outward, but with the rough test she apparently had a vertical diplopia. Both pupils were dilated; the right pupil was slightly larger than the left. The veins of the eyes were full, but the margin of the disks was distinct. The neck was slightly stiff and there was a slight Kernig sign. There was a suggestion of a Babinski sign in the left leg. The pulse was very slow; it had been as slow as 50.

The diagnostic impression was that "the child had a deep suppurative lesion near the midline. The condition may have been localized meningitis, but it was probably a brain abscess involving the mesencephalon. I think that she had a septic infarct at the time when she fainted, February 25 (three weeks before examination), on the day that she first had an earache. The outstanding symptoms through the disease had been (1) slight sepsis, (2) severe headache, (3) involvement of the second branch of the trifacial nerve, (4) diplopia (vertical) and (5) signs of increased intracranial pressure. There may have been a suppuration of the petrous tip."

On March 18, the child was transferred to the Newark Eye and Ear Infirmary.

For the three days that she was under observation, the pulse ranged from 58 to 64, the temperature from 98.4 to 100.4 F. She had repeated attacks of such severe pain in her temple and at times in her face, teeth, neck and frontal region that morphine had to be administered. She was nauseated, and once she vomited. She was restless and perspired at night. The diplopia was demonstrated to be due to a slight weakness of the left external meatus. The veins of the fundi were dilated, but the margin of the disks were distinct. She had the vestibular manifestations of increased intracranial pressure (the left ear gave no reaction to the

cold caloric test when she was in the upright position, but slight horizontal nystagmus when she was in the recumbent position;[59] there was no dizziness and no past-pointing. She could hear with the left ear. Examination of the blood showed: 12,400 leukocytes, 70 per cent polymorphonuclears, 10 per cent of which were band cells and 60 per cent segmented. One blood culture was positive for *Streptococcus nonhemolyticus* after thirty-six hours' incubation.

A roentgenogram showed "intense cloudiness throughout the entire petrous portion of the left temporal bone extending into the petrous apex, which suggested an infection throughout the entire petrous portion.

"The outline of the posterior clinoid process of the dorsum sellae was slightly broken and appeared decalcified" (C. F. Baker).

Operation.—On March 21, ligation of the left common carotid with the fascia lata was performed under local anesthesia. As soon as it was apparent that the vascular occlusion was without deleterious influence on the cerebral circulation, as shown by the absence of sensorimotor symptoms, aphasia or retinal arterial change, ether was administered. Unlocking of the petrous pyramid was performed, and the posterior buttress was removed. The cells in the angle of the petrosal were found to be carious and contained a few drops of pus. The anterior buttress was undercut from behind forward and the floor of the middle fossa lowered from above downward. The temperosphenoidal lobe was elevated and the cortex of the upper surface of the petrous apex was investigated. The bone appeared hard and could not be perforated with a hook.

The observation of a recurrence of a small drop of pus in the lower anterior portion of the mastoid by an assistant (Dr. Richard Swain) led to the discovery of a minute suppurating fistula which extended inward, slightly anterior to the facial canal, along the posterior surface of the petrous. The fine canal was enlarged by converting it into a groove along the posterior surface of the petrous by the removal of its bony covering posteriorly. On reaching the region below the saccus endolymphaticus (which was distinctly seen), the fistula became much enlarged, and through it a considerable quantity of pus escaped. The passage of a probe demonstrated that the fistula extended inward below the internal auditory meatus into a cavity in the petrous apex. The end of the probe was directly below that portion of the anterior surface of the petrous of the middle fossa that I had failed to perforate with the hook. The distance from the plane of the superior semicircular canal to the bottom of the cavity was 4 cm.

After drainage of the cavity in the apex, a nick in the dura in the region of the internal auditory meatus evacuated a quantity of clear cerebrospinal fluid from the posterior fossa.

Postoperative History.—Immediately following the operation, the child had a marked horizontal nystagmus to the right, with a vertical tendency which at times appeared slightly rotary. At intervals she complained of being dizzy. She vomited when her position was changed. (The operative manipulations obviously had disturbed the membranous labyrinth). She had markedly dilated pupils. Her pulse immediately rose to normal.

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59. Eagleton, Wells P.: Decompressions for the Relief of Disturbances of the Auditory Apparatus of Intracranial Origin: Report of Three Cases with a Previously Undescribed Aural Symptom of Increased Intracranial Pressure, Tr. Am. Otol. Soc. **13**:564, 1912; Importance of the Absence of Reactibility of the Vertical Canals to Stimulation of the Cold Caloric, Tr. Am. Laryng., Rhin. & Otol. Soc., 1922.

During the first week she frequently awoke at night screaming and complaining of pain in her head; however, she would promptly go to sleep again. On the third day after operation she had a slight chill, her temperature rose, and she perspired.

Nystagmus and diplopia soon began to subside and both had entirely disappeared on the tenth day. She was discharged from the hospital during the sixth week after the operation.

Postoperative Observations.—(*a*) Evidences in the Blood of Continued Sepsis: The day following the operation, the Schilling test showed: Band cells, 33 per cent, segments, 51 per cent, leukocytes 17,000, polymorphonuclears, 86 per cent. Three weeks later (April 14) her blood count showed: Bands, 41 per cent, segments, 16 per cent, leukocytes 7,000, polymorphonuclears, 57 per cent, small lymphocytes, 42 per cent and basophils, 10 per cent.

(*b*) Lowering of the Intra-Ocular Tension by the Carotid Ligation:[60] Five days after operation, the tension of her left eye was 14 and of the right 18. Ten days later (April 4), tension of both eyes was 16. On the date of discharge (April 28), the tension in both eyes was from 12 to 15 mm.

CASE 4.—*History.*—A. W., aged 7½ years, had abscesses in both ears. One year before operation the discharge from the right ear ceased, but that from the left continued at intervals.

On Jan. 5, 1930, she came to the outpatient department. Her mother stated that the child felt sick and had vomited several times that morning. According to a clinic note, there was "no present ear disease."

On January 24, there was acute otitis media in the right ear, but no discharge.

During March, the child had measles.

On May 15, there was left-sided acute purulent otitis media.

On May 17, she was admitted to the Newark Eye and Ear Infirmary. She now had severe pain in the left ear with profuse discharge and tenderness around the mastoid. She could hear a watch on contact (left ear) and a whisper at five feet. The temperature was 100 F.

On May 18, left mastoidectomy was performed (Dr. B. M. Dearmin and Dr. S. H. Baldwin). The cortex was extremely hard and about one-fourth inch thick. The antrum was deep and filled with granulations and pus. There were diseased cells in the tip, under the sinus and in the perilabyrinthine region. The dura over the tegmen was exposed. Culture from the mastoid revealed a streptococcus which hemolyzed blood slowly.

On May 26, the child was discharged to the clinic.

The child had been confined to bed since leaving the hospital, except when brought to clinic for dressings. The mother noticed the left eye was "dark blue," and had lacrimation. On June 3, the child complained of pain in and around the left eye associated with nausea but no vomiting.

On June 16, she continued to complain of attacks of pain. She had slight sweating attacks and felt chilly two or three times.

On June 17, in the middle of the night (12:30 a. m.), she suddenly awoke screaming with pain in the left eye and ear. An hour later (1:30 a. m.) she became delirious, "ran around the house delirious because of the pain and failed to recognize her mother."

In alarm, the mother brought the child to the hospital at 2 a. m. at which time she was rational and quiet, and had a normal temperature. She remained for two

60. Eagleton, Wells P.: Otitic Meningitis, J. A. M. A. **87**:1544 (Nov. 6) 1926.

days. As nothing abnormal was noted, the midnight occurrence was interpreted as being due to "overeating" and possibly a "stomach ache." On June 19, the child was discharged.

On June 21, she still complained of pain about the left orbit. On the preceding day, the mother noticed that the child could not move her left eye outward. She vomited after breakfast. Her temperature varied from 99 to 100 F. She was readmitted to the hospital.

Examination (S. H. B. and W. P. E.).—There was complete paralysis of the left external rectus. No change was noted at the disk, except that the veins were possibly slightly dilated. There was a suggestion of weakness of the right side of the face. No Kernig or Babinski sign or stiff neck was noted. The mastoid wound still discharged a little. The child could hear with the left ear a loud voice at two feet with the noise box in the right ear; a whisper and a watch tick could not be heard. The Weber test referred to the left ear.

During the ten days the child was under observation, she appeared fairly well most of the time. She had a slight rise in temperature—once it reached 101.3 F.—and her pulse was rather fast, from 90 to 124. At irregular intervals, she complained of pain in and over the left eye and in the left ear, associated with severe pain in the head. The attacks occurred especially during the night, and the following day she slept most of the time. The visual fields showed no apparent contraction.

The red blood cells varied from 3,650,000 to 4,100,000; the hemoglobin from 65 to 80 per cent, leukocytes from 12,000 to 13,000; polymorphonuclears from 57 to 62 per cent; small and large lymphocytes, from 32 to 40 per cent; eosinophils from 2 to 3 per cent; large mononuclears from 3 to 5 per cent; band cells, 5 per cent; segmented forms, 52 to 56 per cent.

In roentgenograms (anterior position), when contrasted with the opposite side, the line of the superior border of the petrous of the left side was irregular beyond the superior semicircular canal. The petrous apex was eroded, with irregular areas of bone formation in the labyrinth and apex regions.

The lateral views showed erosion of the line of the superior petrosal (fig. 3).

Operation.—On July 3, the left common carotid artery with the fascia lata was ligated under local anesthesia, the vessels of the ocular fundus being under direct ophthalmoscopic examination during the whole procedure. The strip of fascia lata was tightened and loosened several times without producing any perceptible effect on the retinal vessels, without alteration of the consciousness or the production of aphasia, hemiphlegia or numbness. The effect of the ligation on the ocular tension (finger test) was immediate. The tension in both eyes fell; they became soft, the tension of the left eye being decidedly lower than that of the right. Ether was administered. Unlocking of the petrous pyramid was performed, also dislocation of the lateral sinus backward and the superior petrosal upward and backward. The anterior buttress was undercut and the floor of the middle fossa lowered. All the bone work was practically bloodless because of the ligation of the carotid.

The dura covering the floor of the middle fossa was elevated. Beyond the prominence of the superior semicircular canal, the superior surface of the petrous appeared rough, eroded and wormeaten. The vessels going through the foramen spinosum were now distinctly seen.

Keeping along the posterior border of the petrous, the posterior edge of the third branch of the trifacial nerve was seen descending toward the foramen ovale. The cortex of the bone under the ganglion was perforated with ease by the hook

which entered a large cavity in the middle of the apex; no pus was seen, although the opening was enlarged. An attempt to "scoop out" the cortex caused rather active arterial bleeding, and I was fearful that injury had been done to one of the vessels surrounding the carotid; however, allowing the dura to fall back in place controlled the bleeding. Moderately tight closure was obtained, and a rubber strip was left along the superior surface of the petrous as far as the opening in the apex.

Postoperative Course.—Following the operation, the child's temperature never rose above 100 F. (rectal), but her pulse rate until the third week continued rather fast, from 100 to 130, only on rare occasions descending to 90.

The drain was removed on the twenty-third day after the operation. The external ocular paralysis still persisted slightly when the child was discharged from

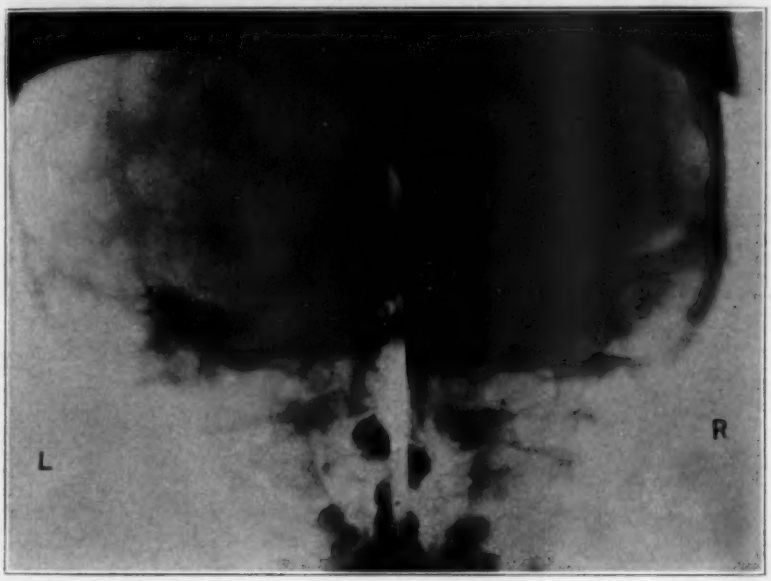

Fig. 3.—Roentgenogram, anteroposterior view (prior to operation) showing erosion of the line of the superior petrosal with irregular deposits of bone in the apex.

the hospital on August 1. The intra-ocular tension continued to be low in the homolateral eye; on July 30, it was 25 in the right eye and about 15 (Schiotz) in the left eye (fig. 4).

Comment.—Because of erosion of the superior surface of the petrous while the interior (medulla) of the apex was soft, I regarded the pathologic process as primarily a local compact bone infection from the veins of the tympanomastoid region, with secondary involvement of the medulla of the apex.

THE SURGICAL TREATMENT OF SUPPURATION OF THE PETROUS APEX BY UNLOCKING THE PETROUS PYRAMID

Cases 2, 3 and 4 were brought to a successful issue largely as the result of a study, during the past few years, of the architecture of the bones forming the base of the skull of the middle fossae, because repeated postmortem examinations of patients dying of suppurative meningitis uniformly had shown that, untreated, the central area of the bulbar (pontile) cisterna is always the seat of a firm exudate. It is apparently from the pontile cisterna that the inflammation ultimately

Fig. 4.—Roentgenogram taken three weeks after operation. Note the complete absence of the superior petrosal border internal to the region of the cochlear. The zone of cellular tissue of the petrous apex is cloudy and denser than the normal (right) side.

extends backward over the cerebellum and upward over the cerebral cortex. The development of the operative unlocking of the petrous pyramid, as I have named it—because after its performance on the cadaver, the addition of a fracture of the inferior wall of the external auditory canal will enable the whole petrous to be pulled out of the base of the skull—has resulted in my performing practically the same bony removal for all suppurative intradural surgical conditions that originate primarily from the ear.

SURGICAL ANATOMY OF THE PETROUS PYRAMID

The temporal bone is the center of the bones which form the middle of the skull. Its petrous portion is a triangular wedge in the base of the skull, dovetailed between the sphenoid in front and the occiput behind. The pyramid is locked in, externally by the bone developed from the tympanic ring and the squama, as well as by the mastoid behind and the condyle of the jaw in front. In the fully developed bone, the external labyrinthine wall of the petrous is approximately 1 inch from the outer surface of the skull.

Functional Development.—The external wall of the labyrinth at birth looks directly outward; the oval and round windows and the facial nerve are very superficially placed, all being about 2 mm. internal to the outer osseous surface.

Likewise at birth, the glenoid surface of the squama is not a cavity but a flat bony plate with a direction chiefly outward and an indentation in its posterior third. During the first year, all the bony structures external to the labyrinth develop outward. The tympanic ring forms the external auditory meatus the anterior wall of which, along with the thickening and the outward movement of the zygomatic portion of the squama, constitutes the "anterior buttress." Coincidently, the appearance of the mastoid cells in conjunction with the posterior arm of the tympanic ring enormously increases and strengthens the bony covering over the sinus. This we call the "posterior buttress."

All this outward bony development is for the protection of the petrous pyramid.

As a result in the fully developed temporal bone the petrous pyramid with its contained labyrinth and facial nerve comes to be placed from ¾ to 1 inch internal to the external bony surface of the skull.

Physiologically, then, the entire outer one third of the temporal bone is simply bony protective superstructure, developed long after birth. It is nature's protection from external violence for the deeper placed neural tissues. Functionally, all this superstructure is unimportant, and can be surgically removed by reversing the mechanism of its development.

Diagnostic and Operative Importance of the Labyrinth.—The labyrinth is anatomically and physiologically the center of the architectural structure of the temporal bone. It and its developmental accessory, the facial nerve, are the chief considerations in all diagnostic speculations and operative manipulations, for when disease from the ear attacks the cranial cavity it enters through the labyrinth, under the labyrinth or over the labyrinth or by way of the venous sinuses and veins surrounding the labyrinth.

The Functional Purpose of the Widening of the Floor of the Middle Fossa.—In the development of the skull after birth, the floor of the middle fossa becomes widened; this is to allow continued growth of the cerebral tissue of the temporosphenoidal lobe, especially of the association fibers that are situated directly behind the pterion. The latter are among the latest additions to the cerebral architecture. It was through their evolution that man was lifted above the animal through the acquisition of speech.

For the protection of the cerebral tissue of this area, as well as to permit its continued development after birth, nature availed itself of the phylogenetically ancient anterior buttress of the squama and the flying buttress of the zygoma. For it is the "flying buttress" of the zygoma that protects and at the same time allows a continuation of growth of the cerebral tissue of the temporosphenoidal lobe through nonclosure of the circummeatal sutures, especially those forming the pterion.[61]

Protection of the Petrous by the Glenoid Fossa and the Condyle of the Jaw.—The outward growth of the tympanic ring and the tympanic portion of the squama with its contiguous zygomatic roots, carry the mandibular fascet (which at birth is well in front of the petrous) inward and backward so that the fully formed glenoid fossa, as well as the articulation of the lower jaw, come to lie over the anterior lower one third of the petrous, while the zygomatic roots of the squama (which at birth are far in front of the petrous) now cover the anterior and superior two thirds of the labyrinth—the region of the cochlear.

The technical problem in suppuration of the petrous apex is how to expose the petrous apex in the floor of the middle fossa and in the posterior fossa without injury to the labyrinth, the facial nerve, the sinus and the carotid artery and still preserve that mobility of the jaw which is necessary for mastication.

Anatomically, (1) the facial nerve surrounds the petrous behind and above. (2) The carotid artery is within the petrous. (3) Externally, the articulation surface of the lower jaw is over the anterior one third of the petrous below, while the upper surface of the glenoid fossa (anterior buttress of the zygoma and the squama) covers the anterior and upper two thirds of the petrous. (4) The sinus is behind, and (5) the jugular bulb is below.

Pathologic Indications in Suppuration of the Apex.—An exposure is called for which will allow direct inspection of the four routes by which the petrous apex is known to be infected: (1) through the labyrinth when the route of infection is by (*a*) direct extension of the suppurative process or by a venous channel from the middle ear through

61. Harris, H. O.: Closure of the Cranial Sutures in Relation to the Evolution of the Cerebral Cortex, Bull. Mayo Clinic, Aug. 31, 1927.

the labyrinth [62] or when (*b*) the labyrinth is secondarily infected from suppuration in the apex, which follows a thrombophlebitis of the veins surrounding the labyrinth [50] (case 2, McE., former series, and case 1, M. G., present report); (2) by way of the peritubal cells (possibly case 1, A. R., former series); (3) by a thrombophlebitis of the veins along the superior surface of the petrous (case 2, M. L.[63]), and (4) by the suprajugular cells (case 3, G. D.).

SURGICAL OBJECTS IN THE OPERATIVE UNLOCKING OF THE PETROUS PYRAMID

1. *Exposure of the External Wall of the Labyrinth and Eustachian Tube Region to Unobstructed Inspection.*—This exposure enables the surgeon, by the sense of sight, looking from above and exteriorly, (*a*) to enter the labyrinth directly or (*b*) to follow the disease through the peritubal cells, when this has been the course of the infection.

Direct unobstructed inspection of the labyrinth and peritubal region is obtained by the removal of the anterior buttress, the tegmen of the antri tympanum and of the hypertympanic space and the overhang of the cells above the eustachian tube when the latter are present.

2. *Elevation of the Dura.*—In cases in which the marrow of the petrous tip is infected and the labyrinth is still functioning, the subsequent elevation of the dura permits exploration of the superior surface of the petrous apex under the gasserian ganglion. (*a*) When a collection of pus is situated here (Bircher's [54] and Streit's [64] cases), it may be evacuated; (*b*) when the bone is the seat of an osteomyelitis with little or no pus formation, the cortex may be widely opened (case 2, M. L.), or (*c*) when the superior surface is eroded and the apex is carious (case 4, A. W.) both may be drained.

Sufficient elevation of the dura of the temporosphenoidal lobe to reach the apex properly is made possible by extensive removal of the squama.

3. *Exposure of the Anterior Surface of the Posterior Fossa.*—This exposure is obtained by (*a*) the sacrifice of the posterior buttress, (*b*)

62. Zange: Vascular Relations Between the Middle Ear and Labyrinth Through the Bony Labyrinth Wall, with Some Comments on the Finer Histological Changes in the Window Membranes and a Demonstration of Histological Specimens, Verhandl. d. deutsch. otol. Gesellsch. **21**:206, 1912. Zange microscopically demonstrated the possibility of a blood stream invasion from the middle ear to the labyrinth by the blood vessel canal in the bone running from the niche in the oval window to the ampulla of the posterior semicircular canal.

63. Siebenmann, F.: Korrosions—Anatomie des knochernen Laybyrinthes, Wiesbaden, 1890.

64. Streit, H.: Weitere Fälle endocranieler Complication im ocular und chirurgischen Fall 15. Mittelohreiterungen, Arch. f. Ohrenh. **56**:181, 1902.

the freeing of the sinus and (*c*) the removal of all the bone of the "angle of the superior petrosal sinus," so named by H. C. Barkhow, inward to the junction of the superior semicircular and the posterior semicircular canal.

4. *Reflection of the Sigmoid Sinus Slightly Backward.*—Sufficient bony removal of the mastoid and superior petrosal regions permits reflection of the sigmoid sinus slightly backward. This allows (*a*) direct inspection of the dura covering the anterior surface of the cerebellum and (*b*) of the bone of the posterior surface of the petrous as far as the niche of the saccus endolymphaticus. It will reveal (*c*) any fistula or collection of pus coming from the posterior or superior semicircular canals or (*d*) in the region of the saccus endolymphaticus (a frequent cause of localized meningitis in my experience). In the inferior part of the wound it will expose (*e*) the narrow line of cells, occasionally present, that extend inward from the inferior mastoid region within the petrous beneath its posterior surface, passing below the inferior arm of the posterior semicircular canal, between it and the jugular bulb. The most external of these cells are covered by the facial nerve—the compact canal of which completely overlies them toward the styloid foramen.

Without the complete exposure of the posterior fossa (by the reflection of the sinus and the removal of the angle of the superior petrosal, with the reflection of the sinus and dura backward as described), this chain of cells will be overlooked (as occurred in the early part of the operation in case 3).

5. *"Uncapping" of the Mouth of the Minute Tract.*—When these "digastric groove jugular bulb cells" are the course of the infection into the petrous apex, the operative exposure of a minute opening in the inferior part of the wound behind and internal to the facial canal will allow of the "uncapping" of the mouth of the minute tract by removal of the firm cortex of the posterior surface of the petrous pyramid. A groove is thus formed that passes below the posterior semicircular canal and above the jugular bulb and continues inward between the saccus endolymphaticus and the internal auditory canal above, and the jugular bulb and the aqueductus cochlearis below. The groove, after passing the plane of the internal auditory meatus, becomes in case 2 (G. J.) a canal in the bone which leads directly into the medullary substance of the petrous apex.

TECHNIC OF OPERATION OF UNLOCKING THE PETROUS PYRAMID[65]

The operation has developed from the radical mastoid operation, with a modification of the gasserian ganglion operation extension upward, and an exten-

65. Eagleton, Wells P.: The Cerebrospinal Fluid as an Aid to Diagnosis and Treatment of Inflammatory Diseases, J. Laryng. & Otol. **44**:734 (Nov.) 1929.

sion backward to expose the jugular bulb. My associate, Dr. John V. Bissett, has made important contributions to the technical procedures of the routine of removal of both the anterior and the posterior buttresses.

Incision.—The primary incision extends from the top of the attachment of the tragus above to the tip of the mastoid below (fig. 5). The extension upward is about 2½ inches in length and runs well above the squamoparietal suture. It is continued downward in front of the anterior attachment of the auricle toward the zygoma about ½ inch. The extension backward passes from the tip of the mastoid toward the inferior curved line for about 2 inches, parallel with the base of the skull.

Freeing of the Skin.—In freeing the skin from the fascia, the natural lines of cleavage of the anatomic structure are followed without unnecessarily opening muscular planes. The temporal fossa extension skin incisions over the mastoid

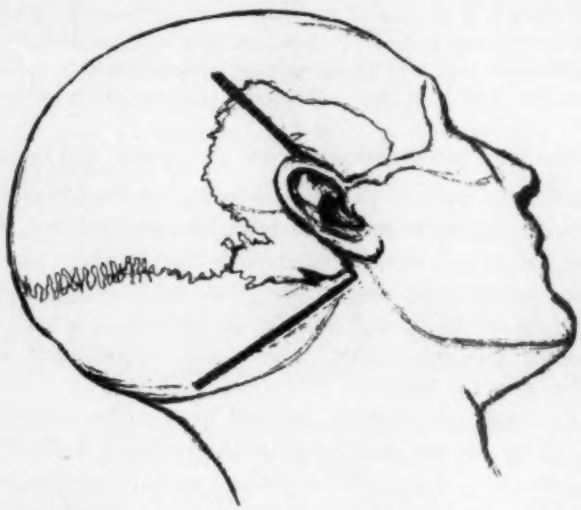

Fig. 5.—Technic of operation of unlocking the petrous pyramid, showing the inferior incision along the inferior curved line of the occipital extending backward 2 inches from the tip of the mastoid.

go through the anterior and external auricular muscles but are carried down to the temporal fascia. In other words, the ear and skin and all the adjacent structure are separated from the fascia of the temporal region above and the sternocleidomastoid below, without disturbing the fascia.

Reflection of Ear Downward and Forward.—The fascial layer is separated from the auricle behind and the facial fibers loosened above. The ear and external auditory canal are now forcibly reflected downward and forward. Provided the fascial covering of the temporal ridge is free but not injured, an incision along the temporal ridge backward and upward will allow the posterior part of the temporal muscle (still covered by the fascia) to be reflected upward and forward, and the covering of the mastoid region backward. This is because the temporal ridge facial layers are attached directly to the bony prominences, while the temporal muscle above springs from the fascia.

In reflecting the mastoid fascia, which is here the periosteum, it peels off easily until the line of attachment of the sternocleidomastoid is reached where its tendon is adherent to the beginning at the inion behind, running downward and forward to the anterior border of the tip in front.

Removal of the Bone for Exposure of the Dura.—Instruments: All removal of bone must be accomplished by broaches rotated by a powerful electrically driven motor (Martley and Kenyon,[66] or the later Albee). There should be at least three sizes of broaches, and at least two motors with aseptic shells, so that time is not lost in changing the broaches and to provide for mechanical accidents to the motor.

Removal of Bone: The broaches must be sharp, and the bone constantly flooded as a dull broach generates great heat by friction against the bone, and when the layer of bone is thin, especially if it is adherent to the sinus or dura, the latter may be burned and a local necrosis result. (This has happened in three instances in the experience of the staff of the infirmary.) A burn may ultimately result in superficial dural or sinus necrosis, which in the presence of surrounding suppuration, may lead to extensive gangrene, although at the time of the burning there may have been little macroscopic evidence of the injury.

SURGICAL ANATOMY OF THE POSTERIOR BUTTRESS

Posteriorly, the petrous pyramid is locked to the bones of the skull by the union of the squama, the parietal, the mastoid and the occipital bones. The posterior buttress is formed by the junction of the superior and posterior surfaces of the petrous pyramid internally, while on the external surface of the skull the posterior buttress is where the petrous pyramid passes into the parietal above, the squama and the mastoid process below and the occipital behind.

Externally, the root of the posterior buttress (the removal of which is the surgical key to the unlocking of the pyramid behind) is formed by that portion of the compact bone that encases the pyramid and divides the middle from the posterior fossa at an obtuse angle and where the petrous joins with the parietal, squama, mastoid and occipital bones.

The posterior extremity of the root of the posterior buttress in a general way lies behind the asterion and directly above the sinus at its knee, the position of the sinus dictating the angle at which the compact tissue of the angle of the superior petrosal joins with the external bones of the skull. Consequently, the farther forward the sinus, the more acute is the angle, a posteriorly lying sinus allowing the root to extend much farther backward.

The internal triangular space between the two compact bony layers of the "angle of the superior petrosal" is called Trautman's triangle. It is filled with cancellous tissue. Cancellous tissue thus normally surrounds the antrum and extends inward for a variable distance on to the pyramid. The extent of the petrous cells depends on the position of

66. Hartley and Kenyon: Experiences in Cerebral Surgery, Ann. Surg. **45:** 481, 1907.

the sinus and the degree of pneumatization of the bone. However, cancellous tissue always fills the space between the horizontal and the superior semicircular canals and the posterior canals in the upper part of the angle of the superior petrosal. This cancellous tissue is usually fat-filled diploic space, but it may be a pneumatic ingrowth from the antrum.

Except when the jugular bulb is low or is placed well forward, there are cancellous spaces in the lower part of the posterior surface of the petrous pyramid. These also are usually pneumatic cells which have grown in from the inner surface of the digastric groove area of the mastoid (digastric jugular bulb cells).

Operative Location of the Root of the Posterior Buttress.—Surgically, to find the posterior end of the root of the posterior buttress (and thus to allow of the exposure of all the cancellous tissue of the buttress, and the reflection of the sinus backward), all the bone of the mastoid and the region posterior and above the knee of the sinus must be removed, as the root invariably covers the knee of the sinus, including its posterior border.

SURGICAL ANATOMY OF THE ANTERIOR BUTTRESS OF THE PETROUS PYRAMID

The anterior border of the petrous portion of the temporal bone is locked into the base of the skull by the great wing of the sphenoid anteriorly and by the squama, zygoma, the glenoid fossa and the lower jaw externally.

In the region of the middle ear the outer labyrinthine wall of the petrous has a direction downward and forward, the cochlear of the labyrinth being deeper placed than its posterior, vestibular area.

That portion of the labyrinth that lies in front of and below the facial nerve is protected by compact bone, formed from the junction and overgrowth of the anterior arm of the tympanic ring below, the glenoid fossa in front and the zygomatic root above.

Overlying the upper and anterior angle of the middle ear, a firm buttress of compact bone for the protection of the labyrinth is developed by the glenoid fascet being carried inward and the root of the zygoma backward. Into this compact mass is dovetailed the anterior external portion of the petrous. It is this dovetailed junction—made up of the tympanic ring, the internal arm of zygomatic process of squama (as the latter passes inward to form the posterior wall of the glenoid fossa) with the anterior wall of the external auditory canal and the floor of the middle fossa—that is called the "anterior buttress."

This compact bone area is doubly triangular, the base of one triangle pointing directly outward, while that of the other points backward.

The anterior buttress completely covers the upper and anterior two thirds of the bony cochlear and the hypertympanic space of the middle ear.

Below and internal to the anterior buttress, that portion of the glenoid plate which is developed from the anterior arm of the tympanic ring forms the anterior and lower wall of the osseous eustachian tube.

Although the triangular, anterior buttress cannot be entirely removed as it supports the condyle of the jaw, a V-shaped area of its anterior meatal wall area can be undercut in it from behind, and its cranial surface lowered from above downward without injury or weakening of the buffer to the joint.

Operative Effect of Undercutting the Anterior Buttress, the Removal of the Tegmen and the Elevation of the Dura.—If the posterior triangular base of the anterior buttress is brazed from behind forward to the junction of the internal arm of the zygomatic process with the tympanic ring, the bone of the tegmen tympani ronguered away, and the dura over the temporosphenoidal lobe elevated, the operator views, from above, the promontory, oval and round windows and can see down the tube for several millimeters. He can now by the sense of sight directly open the outer labyrinthine wall, beyond which is the cochlear and the internal auditory meatus

RACE AND SEX VARIATIONS IN AUDITORY ACUITY*

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The causes of the variations in auditory acuity that are seen in the results of routine audiometric tests of hospital patients with "normal" hearing have been studied from the point of view of age [1] as well as from that of the effects of certain systemic conditions.[2] The purpose of the present study is to analyze the differences shown in the results secured by tests of males and females and of white and colored patients.

The records analyzed are, as in the previous studies, secured from tests of patients in the medical and surgical departments of the Johns Hopkins Hospital. All tests were made with the same 1*A* Western Electric audiometer. The testing was done in the wards or in a small room set apart for that purpose. Neither was ideal for testing hearing, as neither was free from the disturbing effects of accessory noises, but the two conditions were quite similar, and patients tested in both places gave results that coincided within the limits of the attention factor. The tests were done by four examiners whose methods and technic were constantly supervised.

No record was used in this study if the patient's complaint was primarily of ear trouble, or if gross defects were shown in the audiograms of either ear. There is one exception to this. When elderly patients are questioned about their auditory acuity, they frequently at first deny any loss, but when questioned further often state that "while my hearing may not be as keen as it was when I was young, it is as good as that of any one of my age." Records from such patients are included; in fact, many of the records used in the present study have been used in the previous studies.

The solution of the problem of racial and sexual variations first necessitated the grouping of the records according to age and redividing them according to race and sex. The accompanying table shows the distribution of patients in the various groups.

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* From the Otological Research Laboratory, the Johns Hopkins University.

1. Bunch, C. C.: Age Variations in Auditory Acuity, Arch. Otolaryng. **9**:625 (June) 1929.

2. Bunch, C. C.: Further Observations on Age Variations in Auditory Acuity, Arch. Otolaryng. **13**:170 (Feb.) 1931.

A statistical comparison of the two races may lead to inaccurate interpretations, since the number of white patients is more than three times as great as the number of colored patients. We have used all the available records, however, and the possibility of statistical errors is

Distribution of Patients According to Age, Race and Sex

Age	White Male	White Female	Colored Male	Colored Female	Total
20 to 30	73	50	17	21	161
30 to 40	63	62	28	21	174
40 to 50	75	51	30	17	173
50 to 60	61	39	22	13	135
Over 60	28	22	..	..	50
Totals	300	224	97	72	693
	524		169		

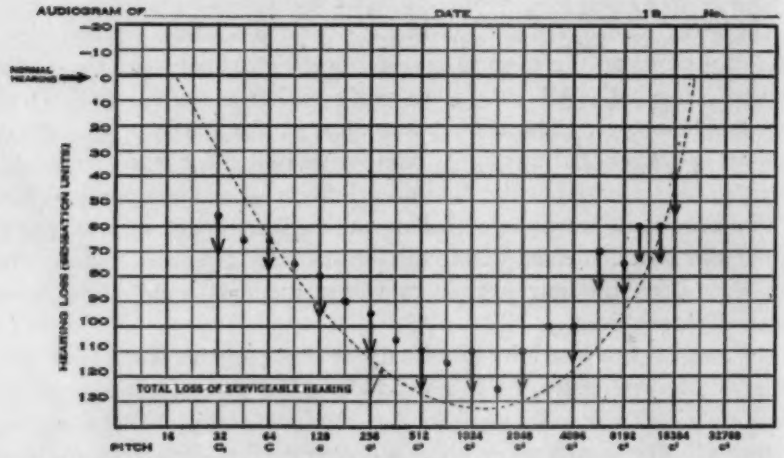

Chart 1.—Audiogram of the Western Electric 1*A* audiometer. The maximum intensities of the tones produced are indicated by dots. The arrows indicate the tones used as stimuli in this study.

recognized. It will also be noted that there are no records for patients under 20 years of age and none for colored people over 60 years of age. Too few records were available to permit an analysis of the hearing in these groups.

Chart 1 is an audiometer chart on which is recorded in heavy dots the tones that are produced by the 1*A* audiometer, the octaves and semi-octaves of *c* from 32 to 8,192 double vibrations and three tones in the next higher octave, viz., 10,321 double vibrations, 13,004 double vibrations and 16,384 double vibrations. The arrows indicate the tones used as stimuli in these tests. The curve of dots on this audiogram indicates the maximum intensities produced by the audiometer. The intensity

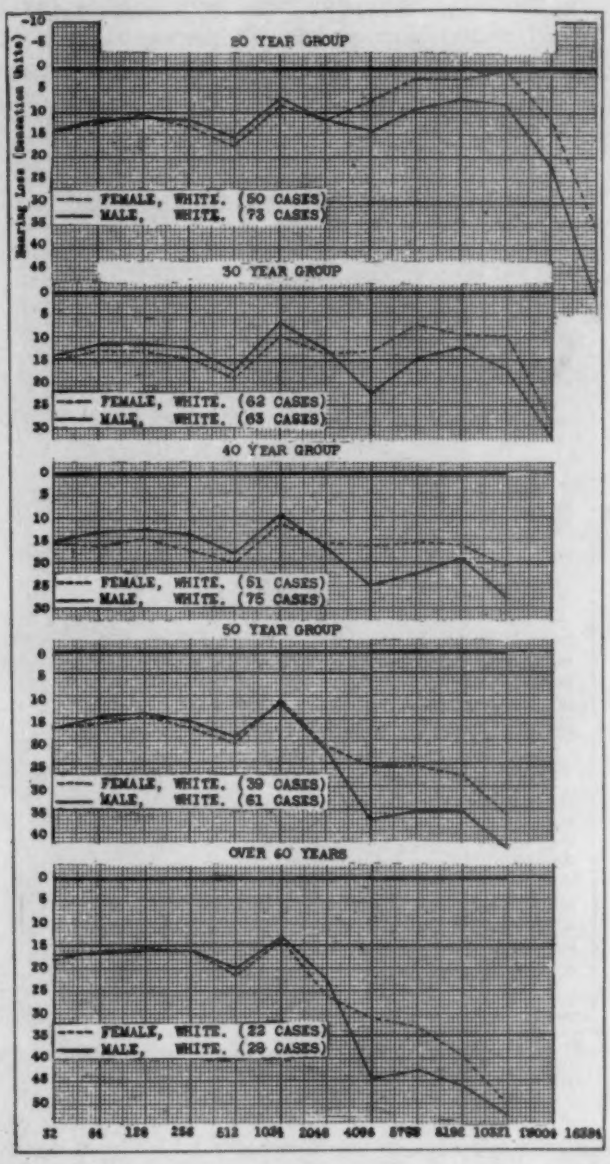
Hearing Loss (Sensation Units)
20 YEAR GROUP
FEMALE, WHITE. (50 CASES)
MALE, WHITE. (73 CASES)
30 YEAR GROUP
FEMALE, WHITE. (62 CASES)
MALE, WHITE. (63 CASES)
40 YEAR GROUP
FEMALE, WHITE. (51 CASES)
MALE, WHITE. (75 CASES)
50 YEAR GROUP
FEMALE, WHITE. (39 CASES)
MALE, WHITE. (61 CASES)
OVER 60 YEARS
FEMALE, WHITE. (22 CASES)
MALE, WHITE. (28 CASES)
32 64 128 256 512 1024 2048 4096 8192 16384

Chart 2.—The averages of the thresholds for white females compared with those for white males. In each decade low tones are heard better by the males, and high tones are heard better by the females.

dial of the audiometer is so constructed that different maxima are produced for different tones. For example, 1,024 double vibrations has 110 "sensation units" for its maximum intensity, 4,096 double vibrations can be produced at 100 units and 16,384 double vibrations at but 40 units. Reference will be made to this variation in the description of the charts which follow.

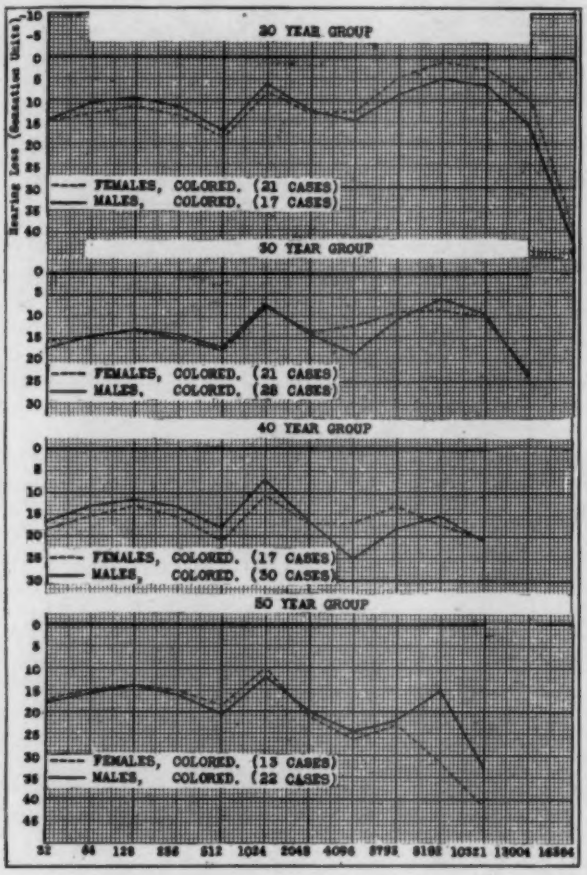

Chart 3.—The averages of the thresholds for colored females compared with those for colored males.

The calculated averages of the thresholds taken from the audiometer records of white males and females are shown in chart 2. In this chart and in those which follow, the pitch of the tones used as stimuli is indicated in the horizontal line at the bottom of the chart, and the intensity average of the thresholds is indicated in the vertical columns at the left.

The curves for white males and females lie close together up to the tone 1,024 double vibrations, that for the males being somewhat closer to the indicated normal (marked 0 on the chart) than that for the females. This is true for all tones in all age groups except 32 double vibrations and 128 double vibrations in the 20 year group, 32 double vibrations and 1,024 double vibrations in the 50 year group, and 64 double vibrations, 128 double vibrations and 256 double vibrations in

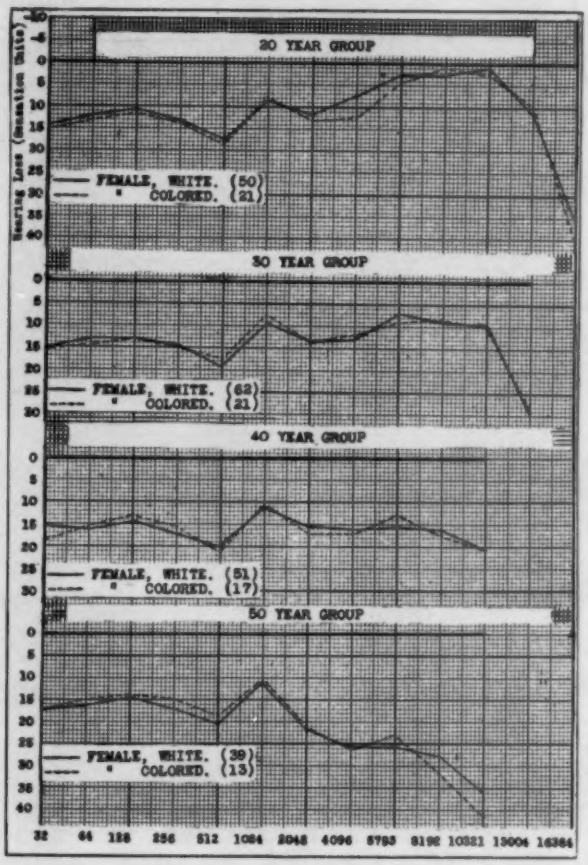

Chart 4.—The averages for white and colored females.

the 60 year group. The difference between the averages of the males and females for the tones under 2,048 double vibrations is not great, but the agreement of the curves for all ages is striking and indicates that men hear low tones better than women.

The most surprising and unexpected point brought out by this study is that between 1,024 and 2,048 double vibrations the curves cross. The females in all age groups hear high tones better than the males. The

difference for the averages of the thresholds for the two sexes is distinct in all age groups, that for females being distinctly nearer the indicated normal than that for the males. In all except the 20 year group the greatest difference is at 4,096 double vibrations.

The averages for thresholds for colored males and females are shown in chart 3. In the 20 year group, low tones are heard best by the males

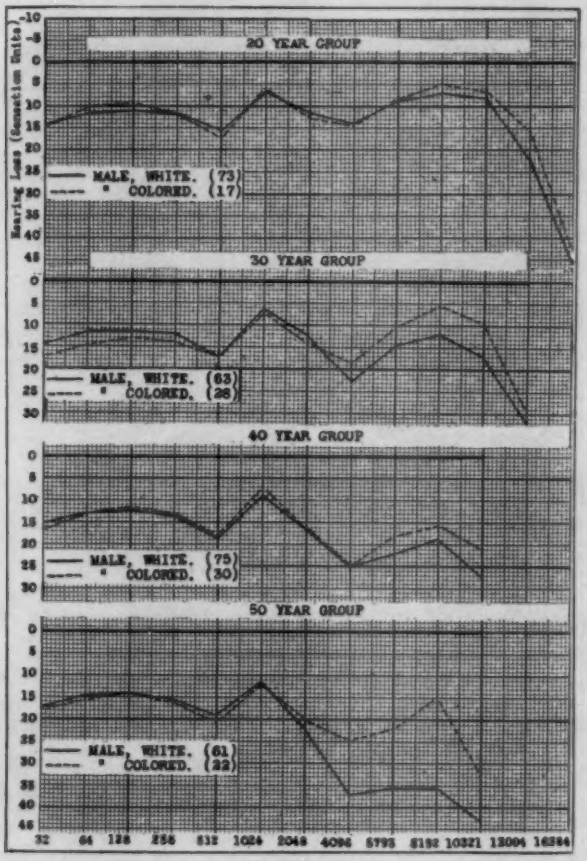

Chart 5.—The averages for white and colored males.

and high tones by the females, but this difference is not consistently shown for the other age groups. There is, however, a constant dip for 4,096 double vibrations for all groups of males, and the fact that this is not true for females suggests that this impairment for 4,096 double vibrations is due to an organic lesion somewhere in the auditory apparatus that is present in men more commonly than in women. It certainly cannot be explained by a mechanical defect in the audiometer.

The dip at 512 double vibrations followed by the rise at 1,024 double vibrations is constant for all ages and sexes, both white and colored, and is probably due to the mechanical construction of the audiometer and the conditions of the test.

The comparison of the average thresholds of white and colored females is shown in chart 4. The uniformity of the curves is striking,

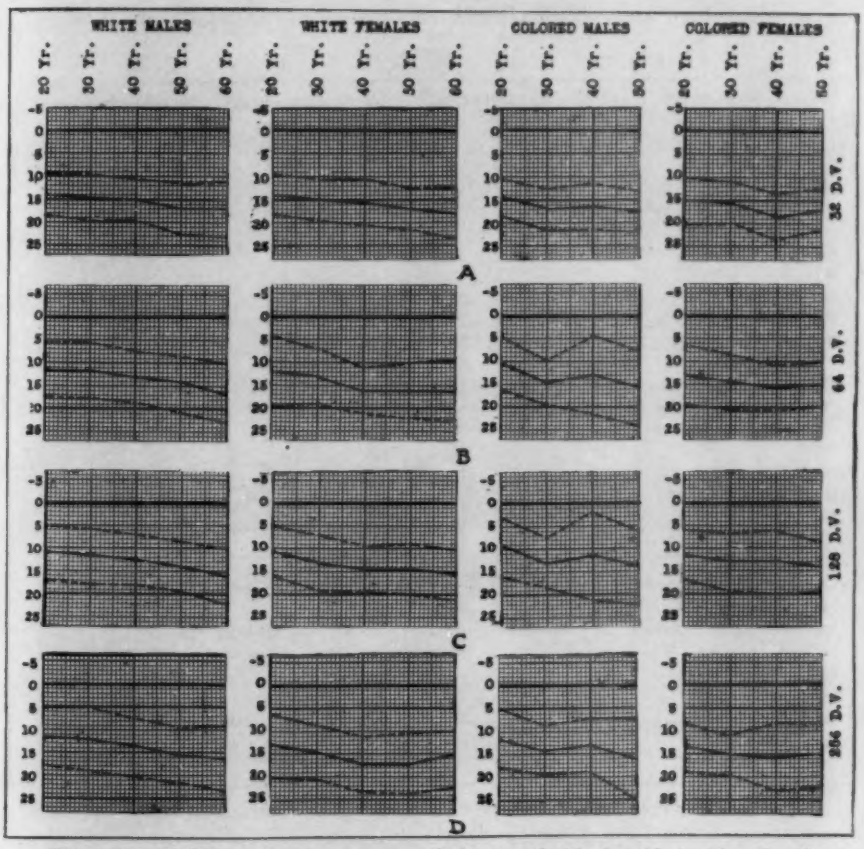

Chart 6.—*A*, changes in the average of the thresholds for 32 double vibrations which appear in successive decades. The solid line indicates the averages, and the lines of dashes, the average variations; *B*, for 64 double vibrations; *C*, for 128 double vibrations and *D*, for 256 double vibrations.

although there are almost three times as many white as there are colored females in each age group. The curves in chart 4 are in marked contrast with those in chart 2.

Chart 5 shows a similar comparison for white and colored males. The average for low tones for colored males is below that for white only in the 30 year group, but the average for high tones is consistently

better for colored males in all age groups. In the 50 year group, the dip in at 4,096 double vibrations is constant in both races.

In each of the charts so far presented the average is computed for the tone 16,384 double vibrations in only the 20 year group. In the 30 year group this tone is not computed, and in the 40 and 50 year groups the tone 13,004 double vibrations is not computed. This variation in the curves is due to the fact that few in the more advanced age groups hear these high pitched tones even at the maximum intensity

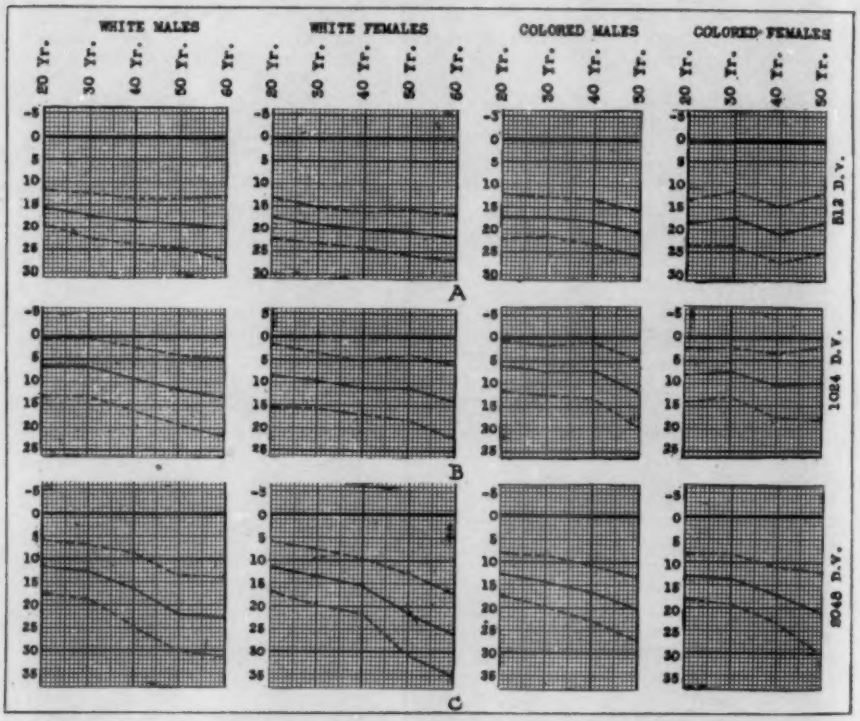

Chart 7.—*A*, changes in the average and average variation which appear in successive decades for 512 double vibrations; *B*, for 1,024 double vibrations and *C*, for 2,048 double vibrations.

produced by the audiometer. In the 30 year group, few hear 16,384 double vibrations at its maximum intensity (40 sensation units), and few in the 50 year groups hear the tone 13,004 double vibrations at its maximum (60 sensation units). For this reason the thresholds for these tones are not averaged in the chart.

The race, sex and age changes in acuity for each octave from 32 to 8,192 double vibrations were plotted to the audiometric scale and are illustrated in charts 6, 7 and 8. In each chart a broken solid line indicates

the means and the line of dashes the mean variations. For the tone 32 double vibrations there is less than five units' (the smallest division of the intensity dial) difference between the averages for the 20 year

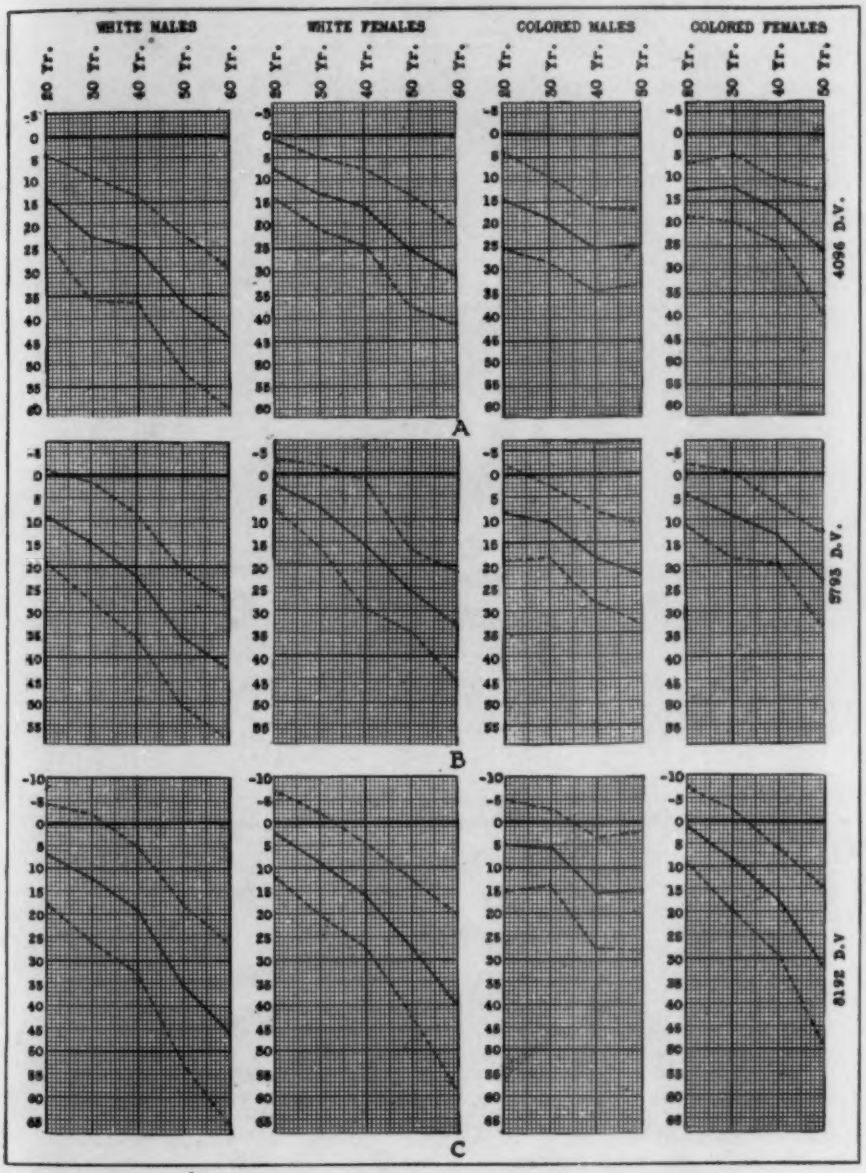

Chart 8.—*A*, changes in the average and average variation which appear in successive decades for 4,096 double vibrations; *B*, for 5,793 double vibrations and *C*, for 8,192 double vibrations.

and the 60 year groups of both races. For 64, 128, 256 and 512 double vibrations, the change is approximately five units for each race and sex. At 2,048 double vibrations and for tones of higher pitch the impairment

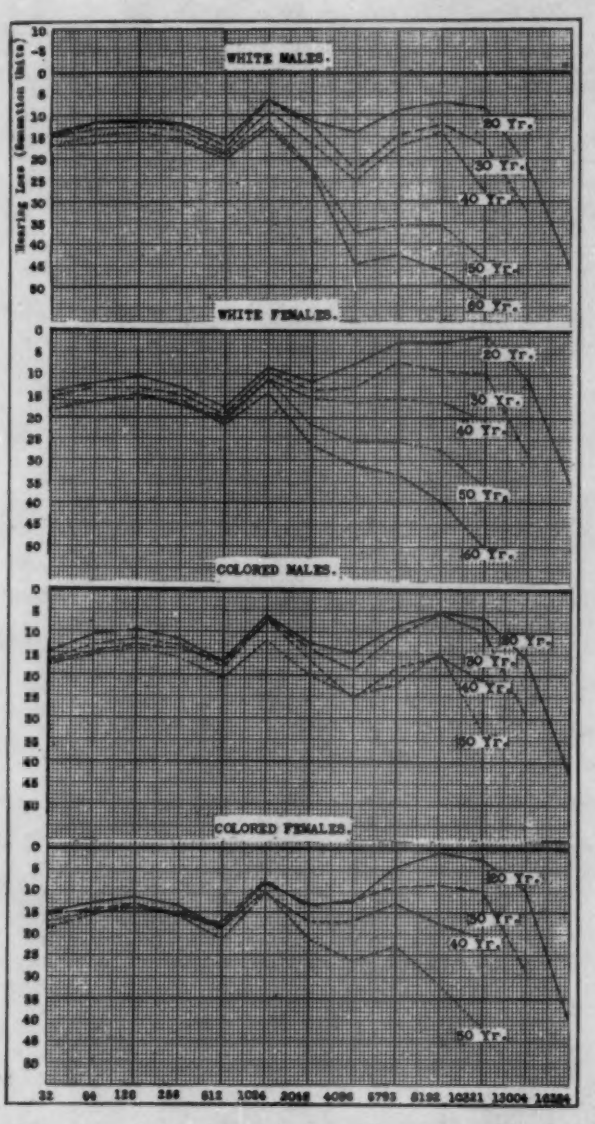

Chart 9.—The means for each decade for white males, white females, colored males and colored females.

is shown clearly. It is interesting to note that white males from 20 to 60 years of age hear all tones above 4,096 poorer than do any of the other groups (chart 8). It appears less marked for colored than for

white subjects, but it should be remembered that the number of colored patients is less than the number of white, and that no records of colored people over 60 years of age were tabulated.

Chart 9 shows a comparison of the changes occurring with age for all tones and for each race and sex. In each instance, the changes occurring for tones under 2,048 double vibrations are slight. At 2,048 double vibrations the difference is more marked and is greater for tones of higher pitch.

Chart 10 shows the age changes for all groups combined in a single chart. In making this chart all available records were used without differentiating race or sex. It seems definite that the acuity for all tones under 2,048 double vibrations changes little with advancing age, less than one division of the audiometer scale, but since the curves do not cross and touch only at one point (256 double vibrations), the change

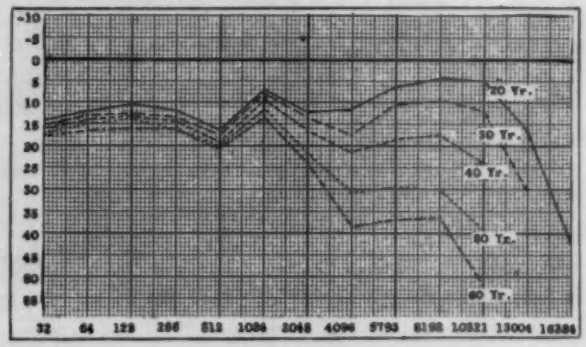

Chart 10.—The means for each decade for all patients regardless of race and sex.

is definite. It was impossible to show this change in the earlier study in which the averages of but 353 cases were studied. In this study of 693 cases it becomes evident. The loss in each decade at 2,048 double vibrations becomes more marked than it is for tones of lower pitch, and it increases constantly for tones of higher pitch.

SUMMARY

The averages of the thresholds of auditory acuity secured by audiometric measurements on patients from the medical and surgical departments of the Johns Hopkins Hospital are computed according to age, sex and race. The average acuity for tones under 2,048 double vibrations is slightly higher, i. e., tones of faint intensity are heard better by white males than by white females. Near 2,048 double vibrations the averages for the two sexes cross, and the average for females is higher (fainter) than for males. This is true for all decades.

The differences between colored males and colored females are not definite. The average for colored males is higher than that for white males for tones of high pitch.

In each successive decade the average of the thresholds is slightly lower (louder) for tones of low pitch. For tones of high pitch, the difference is more marked than for low pitched tones, and the difference between the averages for each age group is greater as the frequency of the tones used as stimuli increases.

PARALYSIS OF THE VOCAL CORDS*

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A brief review of the innervation of the larynx will show that all of the muscles of the larynx, with the exception of the cricothyroid, which is supplied by the superior laryngeal nerve, are supplied by the recurrent laryngeal nerve, often called the motor nerve of the larynx. The recurrent laryngeal nerve, it will be recalled, has its origin in the medulla oblongata on the floor of the fourth ventricle, leaves the cranial cavity in the trunk of the vagus nerve and is contained in this nerve until it has entered the thorax; here the recurrent laryngeal nerve follows a different course on each side.

On the right side it leaves the vagus nerve just in front of the subclavian artery, winds backward around that vessel and ascends obliquely to the side of the trachea behind the common carotid artery, and either posterior to or anterior to the inferior thyroid artery. Fowler and Hanson,[1] in a report on the dissection of 400 nerves, stated that the recurrent laryngeal nerve passed posteriorly to the main branches of the inferior thyroid artery in 65 per cent of the nerves. In 26 per cent it passed anteriorly, and in 8 per cent it passed between the main branches of the artery. On the left side, the recurrent laryngeal nerve arises in front of the arch of the aorta, but winds posteriorly around the aorta to the point at which the remains of the ductus arteriosus are connected with it, and then ascends to the side of the trachea. The nerves on both sides ascend in the groove between the trachea and the esophagus, and, passing under the lower border of the inferior constrictor muscle, enter the larynx behind the articulation of the inferior cornu of the thyroid catilage with the cricoid being distributed to all of the muscles of the larynx except the cricothyroid, as already noted.

In this series of cases there were twenty-seven women and twenty-two men. The youngest was 21 and the oldest 73 years of age, with six between 20 and 30, eight between 30 and 40, ten between 40 and 50, eleven between 50 and 60, thirteen between 60 and 70 and one over 70, making a total of forty-nine cases.

* Submitted for publication, Oct. 4, 1930.

* Thesis submitted to the Faculty of Otolaryngology of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Master of Medical Science for graduate work in Otolaryngology.

1. Fowler, C. Haynes, and Hanson, William A.: J. Am. Coll. Surgeons **49**:59 (July) 1929.

There were five cases of involvement of both recurrent laryngeal nerves and forty-four of involvement of one side only. In the latter, the position of the affected cord was as follows: Twenty were fixed in the midline, three in the outer or expiratory position, eight in the cadaveric position, one just lateral to the midline and twelve the position of which was not stated. The paralysis in twenty-seven involved the left cord, and in seventeen the right.

Of the cases of involvement of both cords, two were due to paralysis of the internal tensor muscles, and while their movements of abduction and adduction were normal, on phonation a bowing of the cords took place. In two cases of carcinoma of the thyroid gland, the

An Analysis of Forty-Nine Cases of Paralysis of the Vocal Cord (or Cords) Due to Interference with the Nerve Supply, Compiled from the Ear, Nose and Throat Department of the Cleveland Clinic *

Causes of Paralysis	Unilateral Cases		Bilateral Cases	Total
	Right	Left		
Previous operative trauma	5	3	..	8
Benign thyroid enlargement †	5	1	..	6
Carcinoma of the thyroid gland	1	2	2	5
Following roentgen treatment of the thyroid gland	1	0	..	1
Intrathoracic goiter	1	3	..	4
Enlarged heart	0	4	..	4
Aneurysm aorta ‡	0	2	..	2
Pulmonary tuberculosis	2	5	..	7
Calcified mediastinal glands	1	2	..	3
Carcinoma of the breast and mediastinal glands	0	1	..	1
Jugular foramen syndrome	0	1	..	1
Cerebral neoplasm	0	0	1	1
No cause after investigation	1	3	2	6
Total	17	27	5	49

* Only cases in which the larynx was free from tumors, infiltrations or perichondrial changes are included in this series.

† Of the six cases of thyroid enlargement, four were adenomatous goiters and two hyperplastic goiters.

‡ Both patients with aneurysm of the aorta gave positive Wassermann reactions.

vocal cords were fixed just off the midline allowing a small airway. In one case, owing to a cerebral neoplasm, the right cord was fixed in the cadaveric position, and there was a bowing on adduction of the left vocal cord.

In this series the Wassermann reaction was negative in thirty-seven cases and positive in two cases, with no report in ten cases.

An analysis of the symptoms in the cases of paralysis of one vocal cord reveals that impairment of the voice, ranging from "cracked" voice to absolute loss of voice, was the outstanding symptom. This symptom was absent in eight cases, and the voice was normal. Dyspnea was present in fifteen cases and was of the type induced for the most part by exertion. Dysphagia, especially for solid food, was present in six cases, coughing in seven cases, a choking sensation in ten cases and marked inspiratory stridor in only three cases.

A study of the causes of the paralyses revealed the following: No cause could be found after thorough investigation for the two cases of paralysis of the internal tensor muscles, and these were attributed to functional disturbance. One case of bilateral paralysis was due to carcinoma of the thyroid and mediastinal glands, another to carcinoma of the thyroid gland and esophagus, and the remaining case to a cerebral neoplasm on the floor of the fourth ventricle. Of the cases of unilateral paralysis, eight were due to previous operative procedures on the thyroid glands, three to carcinoma of the thyroid gland and four to intrathoracic goiters; aneurysm of the aorta accounted for two cases and cardiac enlargement for four cases. Seven were attributed to pulmonary tuberculosis and three to calcified mediastinal glands. One case associated with the jugular foramen syndrome was due primarily to a tumor mass in the nasopharynx, which, invading the brain via the jugular foramen, affected all of the nerves taking their exit from the cranial vault through the jugular foramen on the left side. One case was due to enlarged mediastinal glands secondary to carcinoma of the breast, and in four cases no apparent cause could be found after a thorough and complete investigation. One case followed roentgen treatment of the thyroid gland, and finally, six cases were due to pressure produced by a benign enlargement of the thyroid gland.

During the thirty-eight months covered by this series, 8,000 patients with disease of the thyroid, exclusive of malignant disease and intrathoracic goiters, were given an indirect laryngeal examination in the ear, nose and throat department of the Cleveland Clinic. In these 8,000 cases, 13 cases of unilateral paralysis of the vocal cords were found. Further examination showed, however, that of these 13 cases, 1 had calcified mediastinal glands, 2 had pulmonary tuberculosis, 3 had a markedly enlarged heart, 1 followed roentgen treatment of the thyroid gland and in the remaining 6 no pathologic process other than an enlarged thyroid gland could be found, and so the paralysis of the vocal cord was attributed to pressure from the gland. This makes 1 case of paralysis of the vocal cord to approximately 1,300 cases of benign enlargement of the thyroid gland, an incidence of less than 0.1 per cent. This figure is at great variance with those previously published, but it is in accord with my own experience, as in the course of making laryngeal examinations of several hundred cases of thyroid disease, I saw only 1 case of paralysis of the vocal cord which was produced by the pressure of a benign enlargement of the thyroid gland. Waugh,[2] in 1922, gave the incidence as about 10 per cent, and Barlow,[3]

2. Waugh, J. M., in Crile, G. W., and Associates: The Thyroid Gland, Philadelphia, W. B. Saunders Company, 1922.

3. Barlow, in Jackson, A. S.: Goiter and Other Diseases of the Thyroid Gland, New York, Paul B. Hoeber, Inc., 1926.

in 1926, stated that the incidence was 6 per cent. In 1927, Kelly,[4] while not presenting any figures of his own, quoted Waugh and Barlow. Mullin,[5] however, stated that "the relation of disturbance of the thyroid to the larynx has been overestimated"; he stated further that "a benign enlargement of the thyroid gland does not cause laryngeal paralysis."

Of the 56 cases of malignant disease of the thyroid examined, 5, or almost 10 per cent, showed paralysis of the vocal cord, 2 of these being bilateral and 3 unilateral. Of the 198 cases of intrathoracic goiter examined, 4, or about 2 per cent, showed unilateral paralysis of the vocal cord.

CONCLUSIONS

1. All cases of paralysis of the vocal cord should be thoroughly investigated.

2. Paralysis of the vocal cord is an infrequent complication of non-malignant disease of the thyroid gland.

3. For obvious reasons, a laryngeal examination should precede any operative intervention on the thyroid gland.

4. A diagnosis of laryngeal paralysis should never be made from subjective symptoms alone.

Dr. William V. Mullin and Dr. Justin M. Waugh permitted me to use the material cited.

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4. Kelly, A. B.: Neurological Factors Underlying Immobility of the Vocal Cords, Brit. M. J. **2**:681 (Oct. 15) 1927.

5. Mullin, W. V., in Jackson and Coates: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929, p. 802.

Clinical Notes

THE DISCHARGE OF CEREBROSPINAL FLUID FROM THE EXTERNAL AUDITORY CANAL*

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In searching the files in the library of the New York Academy of Medicine, I have not found any literature on the aural discharge of cerebrospinal fluid other than that following injuries of the head with fracture at the base of the skull. Marcel Rault,[1] in his thesis, gave six case reports of the escape of cerebrospinal fluid from the external auditory canal following injuries of the head with fracture at the base of the skull, and he stated three conditions as being necessary to cause this discharge: "Donc traumatisme extrèmement violent, produisant délaceration de la substance cérébrale, et brèche osseuse"[2] (extremely violent traumatism, producing laceration of the cerebral substance, and a fissured fracture).

The case that I shall report is not a sequel to an injury to the head with fracture at the base of the skull and is, so far as I have been able to ascertain, the first report of a spontaneous cerebrospinal otorrhea.

REPORT OF CASE

History.—P. W., a man, aged 53, came to me on Aug. 11, 1929, complaining of a discharge from the left ear since childhood. Until and for a short time (how long not known) following an operation on the mastoid performed six years before I saw him, the discharge had had a fetid odor, but after the operation it became odorless and intermittent.

About six years before examination, while riding in a street car, the patient had experienced dizziness for the first time. The attack lasted only a few minutes. Some time later, he had experienced a second but more severe attack of dizziness, but he did not recall having felt nauseated or having vomited. Subsequently, a second operation was performed on the mastoid following which the patient felt well, and complained only of a discharge from the ear.

Examination.—Examination of the nose, throat and larynx showed nothing relevant. The cranial nerves, except the left eighth nerve, as will be shown, were normal. The right ear showed an adhesive process with calcification of parts of the tympanum. The left ear showed the scar from an incision into the mastoid with a postauricular depression resulting from the operation. The auditory canal was large and roomy; the posterior wall of the canal was absent, revealing a cavity lined with healthy epithelium—indications of a radical operation on the

* Submitted for publication, Sept. 5, 1930.

1. Rault, Marcel: Études des ecoulements de liquide cephalorachidian et de matiere cérébrale par le conduit auditif et les fosses nasales a la suite des fractures de la base du crane, Faculté de Médicine de Paris, 1912-1913, no. 221.

2. Rault (footnote 1, p. 28).

mastoid. The fundus of the canal contained a clear fluid, which when wiped away clearly showed the cavity of the middle ear.

Because of the history of vertigo, functional tests of the labyrinth, especially of the vestibular mechanism, were made.

The Cochlear Apparatus.—Tuning forks of 64, 128, 256, 1,024 and 2,048 double vibrations were used. Fork *C*, 128 double vibrations, was used for the Weber, Rinne and Schwabach tests, and the following results were obtained:

Right Ear		Left Ear
Lateralized	Weber	
Positive	Rinne	Negative
Normal	Schwabach	Shortened
Shortened	64 double vibrations	1 second
Shortened	128 double vibrations	6 seconds
Shortened	256 double vibrations	Shortened
Shortened	1,024 double vibrations	Shortened
Normal	2,048 double vibrations	Shortened

The Vestibular Apparatus.—Spontaneous nystagmus and past-pointing were absent. Romberg's sign was negative. The fistula test gave positive results; symptoms were occasionally absent, with the slow component on compression toward the opposite side. Cold caloric stimulation of the right ear with water at 60 F. produced normal vestibular responses, whereas similar stimulation of the left ear produced subnormal responses; that is, the nystagmus that was horizontal-rotary appeared after one and one-half minutes of irrigation with cold water, and the duration of the nystagmus was only thirty-five seconds. During the irrigation, the head was held in the optimum position of Bruening. Bárány's rotation test was not done. The Babinski-Weil test[3] showed a deviation of the path toward the left when the patient walked forward and toward the right when he walked backward, indicating a greater preponderance of the right labyrinth over the left.

The long latent period of the nystagmus in caloric irrigation of the left ear indicated a hypo-excitable peripheral labyrinth. A fistula was present in the left labyrinth communicating with the cavity of the middle ear.

The Aural Discharge.—The discharge was seen to be escaping into the antral region of the fundus of the cavity of the middle ear, because the pulsating light reflex was seen in this region. When the discharge was wiped away, the reflex first appeared small and pulsating; then the force of the pulsations diminished pari passu as the size of the reflex increased. Though the discharge appeared to be coming from the region of the antrum, the eustachian tube was thought to be the origin. The eustachian tube was catheterized and air was injected. However, no air escaped into the cavity of the middle ear. To make certain that the catheter was in the orifice of the eustachian tube, the tube was catheterized under direct vision with the aid of a nasopharyngoscope, and air was again injected with similar negative results, indicating conclusively that the eustachian tube was closed off completely from the cavity of the middle ear. Therefore the conclusion was that the discharge from the ear could not be coming from the auditory tube, either from a pathologic condition within the tube itself or via the tube from a pathologic condition of the adjacent tissues, such as the epipharynx, the nose or the nasal sinuses.

Furthermore, the discharge did not come from an acute inflammatory process, as there was no sign of any. Also there was no evidence of a chronic inflammation,

3. Baldenweck, L.: Leçons sur l'exploration de l'appareil vestibulaire, Paris, Vigot Frères, 1928.

no fetid otorrhea; the discharge was clear and watery, not purulent; there was no granulation tissue and no polypi.

Two further possibilities to account for the otorrhea presented themselves: (1) a salivary fistula from the parotid discharging saliva into the cavity of the middle ear and (2) the escape of cerebrospinal fluid into the cavity of the middle ear via a fistula in the labyrinth; as was stated, the fistula test gave positive results. In order to ascertain which of the foregoing possibilities, if any, was acting in this case, some of the fluid from the meatus was pipetted off with a medicine dropper and subjected to chemical tests.

Some of the fluid was tested for saliva as follows: Ten grains (0.65 Gm.) of potassium iodide dissolved in half a glass of water was given to the patient to drink; he was then instructed to chew gum in order to stimulate the salivary flow. The saliva from the mouth was tested at one minute intervals for the presence of potassium iodide, which appeared eight minutes from the time of ingestion. Then, about one half of the fluid (about 3 drops) from the ear was tested for potassium iodide, which was not found. Four minutes later, potassium iodide was still present in the saliva. The rest of the fluid from the ear was tested for potassium iodide, and again it was not found. No trace of potassium iodide was found in the saliva seven minutes after the initial positive test. The foregoing results ruled out the probability of a parotid fistula.

Iodide was tested for as follows: The addition of a few drops of chlorine solution followed immediately by a few drops of starch solution to the fluid tested gave a blue color, indicating the presence of iodine.

At the next visit of the patient, the discharge was tested for cerebrospinal fluid according to the outline given by Hutchinson and Rainey.[4] It was clear, colorless and watery, without odor. It gave a positive reaction for globulin, reduced Benedict's solution and contained no discernible coagulable proteins. Owing to the relatively small amount of fluid obtainable from the ear, it was necessary to collect the discharge on three different visits in order to obtain sufficient fluid. The inference drawn was that the fluid was of cerebrospinal origin, since the foregoing tests were characteristic of this fluid. The results of these tests, together with the clinical picture—absence of demonstrable pathologic changes to explain the usual otorrheas, the unusual character of the discharge, evidence of a fistula in the bony labyrinth and absence of evidence of a salivary fistula—were sufficient to satisfy me that this was a case of cerebrospinal otorrhea.

The pathway that the discharge took in escaping from the labyrinth into the cavity of the middle ear was probably as follows: The escape was made through an opening in the labyrinth communicating with the middle ear, either through the antral portion of the lateral semicircular canal or through an opening made in the fenestra ovalis by accidental dislocation of the stapes, the fluid in the labyrinth being replenished from the cerebrospinal fluid via the aqueductus cochlea, which is a direct channel of communication between the subarachnoid space and the basal whorl of the cochlea.

Treatment.—The rationale of the therapy carried out was to seal the opening of escape from the labyrinth to the middle ear by use of caustics, and so stimulate the formation of granulation tissue and subsequently of fibrous tissue. Silver nitrate (50 per cent) was first tried, and was applied in the region of the light reflex. This treatment was carried out every second day for two weeks with no

4. Hutchinson and Rainey: Clinical Methods, ed. 3, London, Cassell & Company, Ltd., 1923, p. 602.

results. After this a 75 per cent solution was used daily for four treatments with no apparent result. Lastly, the area of the pulsating light reflex was lightly touched with a fused bead of chromic acid, but the patient did not return, and the effect of the chromic acid is therefore unknown.

BIPOLAR ELECTRODE FOR ELECTROCOAGULATION OF THE INFERIOR TURBINATE*

LEE M. HURD, M.D., NEW YORK

The instrument to be described has been in use for about fifteen years. It consists of two terminal needles of spring brass wire, each 4 cm. in length, protruding from an insulated rubber handle. This measures 16 cm. in length. At the proximal end of the handle, flexible conductor cords lead from each wire and are attached to the proper terminals of the diathermy machine.

The instrument is used for the reduction of hypertrophy of the mucous membranes of the inferior turbinate. The inferior turbinate is first anesthetized by means of pledgets of cotton moistened with a suitable anesthetic placed against

Bipolar electrode for electrocoagulation of the inferior turbinate (half size).

the hypertrophied portion and left in place until anesthesia is complete, without the use of epinephrine. The needles of the electrode are now introduced into the submucous tissue by puncturing at the anterior portion of the hypertrophy and carrying the needles into the tissue parallel to the bone of the inferior turbinate. The coagulating current is applied until a white area is seen about the point of emergence of the needles at the site of puncture. This usually occurs in five or six seconds. The needles of the electrode are then withdrawn.

Since each needle acts as a pole of the electrode, the current passes between them only, and it is only this portion of the submucosa that is coagulated. Coagulation begins at the needlepoints and travels backward to the site of the puncture. When properly applied there is no coagulation on the surface of the mucosa, except a line at the site of puncture between the needles.

The method is bloodless and effects a reduction of the hypertrophy of the mucosa without injury to the bone, with a minimum amount of destruction of the mucosa itself. In every large hypertrophies several such punctures may be necessary, either at one sitting or on several different occasions.

39 East Fiftieth Street.

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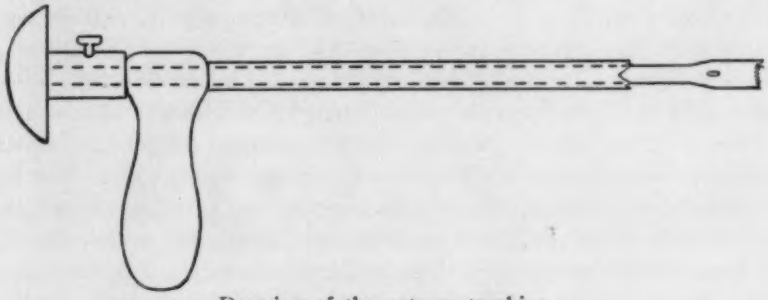
* Read before the Annual Meeting of the American Laryngological, Rhinological and Otological Society, Atlantic City, May 30, 1930.

TREPHINE FOR MAKING A PRETURBINAL OPENING IN THE MAXILLARY SINUS*

H. L. Underwood, M.D., Vancouver, Wash.

I have used a trephine with considerable satisfaction during the past two years in making a preturbinal opening in the maxillary sinus. Two sizes are made by V. Mueller & Company, Chicago, one 7 mm. and the other 9 mm. in size. The accompanying sketch will give an idea of its construction. Apart from the basic essential of a hollow trephine driven by palmar pressure and rotation, the important feature is a sliding metallic sleeve with two deep notches at opposite points on the periphery, which may be applied over the sharp edge of the denuded crista pyriformis just anterior to the lower turbinate. The sleeve is provided with a handle to be held in either the right or the left hand, depending on which antrum is to be opened.

The patient's head is supported firmly against a head rest and turned slightly toward the side on which the operation is to be performed. The instrument is



Drawing of the antrum trephine.

directed somewhat laterally toward the center of the antrum. A few backward turns of the trephine to fray the sharp edge of the bone is desirable in some cases preliminary to the stronger forward cutting pressure. The 9 mm. opening rarely requires the use of a rasp to enlarge it; the 7 mm. drill may do so at times. The remainder of the periphery of the sleeve is sharply toothed, as I employ the instrument occasionally for perforating the wall of the antrum in the canine fossa. For the preturbinal operation only the notches are required. A hole at the base of the trephine facilitates the removal of the button of bone. The instrument could readily be adapted to electric power if desired.

I have found that a large percentage of patients with severe infections of the antrum of nasal origin can be readily cured by the preturbinal operation if the area is thoroughly curetted and drained for two weeks. In dental cases, with probable involvement of the periosteum and bone, however, I do not do the preturbinal operation.

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Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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PERORAL ENDOSCOPY

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In the interpretation of physical signs it is necessary not only to know how to elicit them, but to understand the mechanism of their production.

According to Jackson,[1] bronchoscopic observations, together with correlated studies, demonstrated the mechanism underlying the production of physical signs of foreign bodies in the bronchi, and an understanding of this mechanism was necessary for the proper interpretation of these signs. The studies were greatly aided by checking the physical signs with the bronchoscopic observations of the foreign body and permitting a rechecking of previous interpretations of the physical observations following bronchoscopic removal of the foreign body. The many valuable and practical bronchoscopic contributions to the proper interpretation of physical signs in cases of foreign body led to a similar study of the observations in cases of pulmonary disease of other than foreign body origin. It was observed that viscid or dried secretion, mucosal swelling, granulations and tumors, benign and malignant, at times produced the same phenomena and the same physical signs as foreign bodies.

Because of its fundamental importance in the etiology of pulmonary disease and in the production of physical signs, Jackson gave to bronchial obstruction first place in the analysis of the objective bronchoscopic observations in comparison with the physical signs elicited in the same patient at practically the same time.

Since respiratory air passing through a normal bronchial tube produces no physical sign that is deemed abnormal, Jackson concluded that for the production of abnormal physical signs one or more of four conditions must be present: (*a*) narrowing or enlargement of the lumen; (*b*) swollen mucosa; (*c*) projecting abnormal tissue; (*d*) foreign body, or secretions acting as such (endogenous foreign body). Bronchoscopic observations have determined the fact that there are three different types

1. Jackson, C.: The Mechanism of Physical Signs in Neoplastic and Other Diseases of the Lung: With Especial Reference to Atelectasis and Emphysema, J. A. M. A. **95**:639 (Aug. 30) 1930.

of bronchial obstruction, comparable in action to the three types of valves in mechanics. In the bronchi there may be: (1) stop valve obstruction, in which no air passes in or out of the tributary area of lung, resulting slowly in atelectasis from absorption of the contained air, and later in drowned lung; (2) by-pass valve obstruction, in which a diminished amount of air passes both in and out, and (3) check valve obstruction, in which air is permitted to pass one way only, either in or out, rapidly resulting in emphysema or atelectasis according to the setting of the valvular mechanism. Check valves may be formed by inspiratory enlargement and expiratory diminution of the lumen of infiltrated or inflammatory bronchial walls as well as by mobile tissues or secretions that form ball valve or flapping valve mechanisms.

Alternate expansion and contraction of the thorax in the respiratory cycle may be considered as a reciprocating pump. In mechanics a reciprocating pump may be made to pump air in or out of a reservoir, according to which way the check valve is set. In the check valve type of bronchial obstruction, air may be pumped in or out of the tributary area of lung, resulting in either emphysema or atelectasis, according to the setting of the check valve mechanism. One type of obstruction may follow another, and in some cases two types may alternate.

Full comprehension of the mechanism of each of the three types of bronchial obstruction is necessary for the proper interpretation of the physical signs; when properly interpreted, the signs in the respective conditions become of the utmost diagnostic importance. Bronchial obstruction is so often associated with a normal or hyperresonant percussion note that the utmost care in accurately determining the character of the breath sounds is necessary for exclusion of the minor degrees of the check valve type of bronchial obstruction. Atelectasis in children is so often due to bronchoscopically curable valvular obstruction that the diagnosis of other types of atelectasis should not be made until after diagnostic bronchoscopy has excluded bronchial obstruction.

BRONCHOSCOPY IN DIAGNOSIS

General Considerations.—For practical purposes, Soulas [2] included the indications for peroral endoscopy under several arbitrary groups, as follows: laryngeal syndrome, tracheobronchial syndrome, bronchopulmonary syndrome, bronchial obstruction syndrome, hemorrhagic syndrome and esophageal syndrome. Symptoms commonly observed in these syndromes were enumerated and diagnostic possibilities were discussed. Soulas concurred with endoscopists generally that the results obtained were dependent on many factors. These included an adequate

2. Soulas, A.: Indications for Endoscopic Examination of Air Passages and Esophagus, Presse méd. **39**:651 (May 14) 1930.

instrumentarium, a well trained technical organization, a wide range of practical and clinical experience and cooperation between the physician, roentgenologist, endoscopist and surgeon.

Bronchial Obstruction.—Obstruction of a bronchus may be recognized as such by physical examination combined with roentgen examination. Very often, however, it is impossible to determine the character of the lesion. Funk and I [3] discussed this phase of the question and concluded that bronchoscopy was the only method by which, in a majority of cases, an accurate diagnosis of the cause of bronchial obstruction could be made. This is particularly true when the lesion is endobronchial, as in the case of new growth, granulation tissue, etc.

The importance of the early recognition of bronchial obstruction is obvious. The results of obstruction of a bronchus depend on: (1) the suddenness and the extent to which the entrance and exit of air to the corresponding lung are impeded; (2) the interference with the drainage of secretions from the involved area, and (3) the presence or absence of infection. Long-continued, complete obstruction will lead to pulmonary fibrosis and later to bronchiectasis. Incomplete obstruction often produces obstructive emphysema; in the presence of infection, this commonly eventuates in "drowned lung." The clinical features and some of the problems involved in the diagnosis and treatment were exemplified in a number of case reports.

Pulmonary Atelectasis.—Brunn and Brill [4] agreed that massive collapse and various other types of atelectasis are but different stages of the same condition and that it is the most common postoperative pulmonary complication. Clinical and experimental evidence seemed to favor the theory of bronchial obstruction as the most common single factor in the production of atelectasis. Bronchoscopically, four types of observations were noted: (1) the presence of an obstructing plug or characteristic tenacious sputum, (2) profuse purulent secretion, (3) no secretions in the bronchi and (4) one unusual case of bronchial edema. They expressed the belief that if the condition does not clear up under simple measures, as frequent postural changes or carbon dioxide-induced hyperventilation, bronchoscopy is indicated.

In his observations on cases of postoperative atelectasis which were examined bronchoscopically, Brown [5] confirmed the observations of many bronchoscopists regarding the finding of masses of thick, tenacious secretion. In one patient examined bronchoscopically by him an inter-

3. Funk, E. H., and Clerf, L. H.: Bronchial Obstruction, M. Clin. North America **13**:833 (Jan.) 1930.

4. Brunn, H., and Brill, S.: Observations on Post-Operative Pulmonary Atelectasis, Ann. Surg. **92**:801 (Nov.) 1930.

5. Brown, A. L.: Bronchoscopic Observations in Postoperative Atelectasis: Action of Carbon Dioxide, J. A. M. A. **95**:100 (July 12) 1930.

esting observation was noted; namely, that the right main bronchus was completely occluded by what appeared to be an edematous swelling of the mucous membrane. No thick secretion was observed. Brown observed the effects of the inhalation of carbon dioxide through the bronchoscope and noted that there was an increase in the rate and depth of respiration; this increase in the movements of the bronchial walls tended to free adherent mucus. When a moderate concentration of the gas was administered, there was induced a distinct blanching of the mucous membranes of the trachea and bronchi.

Scheffey [6] and others reported three cases of postoperative pulmonary atelectasis. In two of these, bronchoscopic aspiration of secretion was resorted to with satisfactory results; in the remaining case postural treatment was instituted, with complete relief from symptoms. The writers agreed that the actual cause of the respiratory embarrassment was obstruction of a bronchus. by thick mucus. The predisposing causes were varied, but shallow breathing seemed to be a definite factor. Early change of posture and the encouragement of deep breathing immediately after operation are essential prophylactic measures. Abolition of the cough reflex by narcotic medication must be seriously considered when providing for the alleviation of pain or when prescribing preoperatively. They concluded that while postural treatment should always be attempted, the most gratifying results are those following bronchoscopic aspiration.

Bronchial Asthma.—The occurrence of pulmonary atelectasis as a complication of asthma was discussed by Clarke,[7] and reports of two additional cases, one of them fatal, were added to the literature. It is interesting to note that, in the case in which the patient recovered, a diagnostic bronchoscopy was performed at the time atelectasis was present, but no bronchial obstruction was found. Clarke expressed the belief that lobar atelectasis (massive collapse) occurs in persons with bronchial asthma independent of operative procedures and is frequently fatal. In postoperative atelectasis, previous symptoms of allergy are not uncommon. The fulminating symptoms are suggestive of an allergic reaction to the captured bronchial secretion. Small areas of pulmonary atelectasis are probably not uncommon in persons with true bronchial asthma and may explain some of the severe paroxysms.

Benign Tumors of the Bronchus.—In a review of the literature, Patterson [8] found records of twenty-six cases of benign bronchial

6. Scheffey, L. C.; Jones, H. W., and Clerf, L. H.: Postoperative Collapse of the Lung (Atelectasis) in Gynecologic Cases, Am. J. Obst. & Gynec. **19**:795 (June) 1930.

7. Clarke, J. A.: Pulmonary Atelectasis as a Complication of Bronchial Asthma, Arch. Int. Med. **45**:624 (April) 1930.

8. Patterson, E. J.: Benign Bronchial Neoplasms: Bronchoscopic Aspects, Arch. Otolaryng. **12**:739 (Dec.) 1930.

neoplasms. Of these, sixteen were diagnosed bronchoscopically and ten were found at autopsy. The obvious conclusion is that bronchoscopy affords the only means of making a positive diagnosis of benign neoplasm in the lung. Because of their relatively slow growth, these tumors tend to produce bronchial obstruction and commonly are followed by extensive pathologic changes. In a study of the case reports, it was noted that much confusion existed concerning the interpretation of the symptoms and physical signs.

Primary fibroma predominated in frequency, having been found in eight of the twenty-six cases, and polypoid tumors, next in frequency, occurred in five cases. The bronchoscopic diagnosis of benign neoplasms is not difficult as they appear as smooth, rounded, usually freely movable, pedunculated masses. No difficulty is encountered in securing tissue for biopsy. Bronchoscopic removal of the growths can usually be accomplished without risk of hemorrhage. Large neoplasms may present some difficulty in removal because of dyspnea. Patterson emphasized the importance of early diagnosis of benign neoplasms and their removal by bronchoscopy; if undiagnosed and allowed to remain, extensive pathologic changes will occur.

The diagnosis of adenoma of the bronchus is important because of the favorable prognosis if properly treated. Kramer [9] expressed the belief that these tumors are not so rare as the literature would indicate and that an erroneous diagnosis of carcinoma can readily be made. He reported the cases of two patients who presented obscure symptoms and signs in the chest. A provisional diagnosis of pneumonia or carcinoma was made. At bronchoscopy there was found bronchial obstruction produced by a growth. A provisional bronchoscopic diagnosis of malignant growth of the bronchus was made in each case; however, the diagnosis based on the examination of tissue removed at the time of bronchoscopy was revised in one case to adenoma of the bronchus, whereas the second case was believed to be carcinoma. At a subsequent bronchoscopy in the latter case, additional tissue was removed, and a diagnosis of adenoma was then made. Frequent removal of tissue with implantation of removable radium seeds ultimately led to cure in one case and worked improvement in the other.

Carcinoma of the Trachea.—In discussing the diagnosis of carcinoma of the trachea, Figi [10] stated that this condition, as a rule, is not recog-

9. Kramer, R.: Adenoma of Bronchus, Ann. Otol. Rhin. & Laryng. **39**:689 (Sept.) 1930.

10. Figi, F. A.: Primary Carcinoma of the Trachea, Arch. Otolaryng. **12**:446 (Dec.) 1930.

nized until it is well advanced, in spite of the fact that usually a definite clinical syndrome is produced early. If indirect examination with the laryngeal mirror does not reveal the cause of the trouble and if the symptoms cannot be explained by roentgen and general examination, bronchoscopic study is imperative. In two of Figi's cases the tumor was discovered on indirect examination; in two other cases a diagnosis was made only on bronchoscopic examination, and in the fifth case the condition was not revealed until necropsy.

Carcinoma of the Bronchus.—In discussing the clinical features of neoplasms of the bronchus, McCrae [11] stated that cough is the most common symptom and will occur in practically every case at some time or other. Pain, often localized, is an important symptom and is present in fully 75 per cent of the cases. Hemoptysis, varying from slight streaking of the sputum to severe bleeding, occurs in about 50 per cent of the cases. With the present means of treating patients there are several ways by which the mortality of bronchial cancer may possibly be reduced, namely, by education of the profession, exact diagnosis, intelligent and proper therapy and teamwork. Exact diagnosis often depends on the bronchoscopist, but this chance cannot come unless the profession is more alert to the need for early diagnosis, and can make it, or give the bronchoscopist the opportunity of making it.

In a report on primary bronchial carcinoma, based on an analysis of sixty-one cases, Funk [12] emphasized the value of bronchoscopy in diagnosis and stated that this procedure represented the most recent and important advance in thoracic diagnosis and was the only method by which an early diagnosis of bronchial new growth could be made with reasonable certainty in a vast majority of patients. Without bronchoscopy, the diagnosis often remained in doubt until extrathoracic metastases occurred or tissue was removed at operation or at autopsy. It also has taught physicians to suspect new growth more frequently, particularly when signs of bronchial obstruction are present.

Manges [13] discussed the roentgen observations in cases of bronchial neoplasms and emphasized the point that in any lesion involving change of the tissues in a part of the lung that is accessible to bronchoscopic examination, a final diagnosis should not be made until a satisfactory bronchoscopic study is carried out. With regard to treatment, he expressed the belief that irradiation, even though the results are not

11. McCrae, T.: Bronchial Neoplasms: Clinical Features, Arch. Otolaryng. **12**:727 (Dec.) 1930.

12. Funk, E. H.: Clinical Manifestations of Primary Bronchial Carcinoma, J. A. M. A. **95**:1879 (Dec. 20) 1930.

13. Manges, W. F.: Bronchial Neoplasms: Roentgenologic Aspects, Arch. Otolaryng. **12**:732 (Dec.) 1930.

universally good, probably offers at present the best therapeutic means of combating bronchial neoplasms.

From the point of view of bronchoscopic diagnosis, Jackson[14] divided malignant growths of the lung into three classes: (1) growths primarily endobronchial, (2) growths primarily peribronchial or parenchymal and (3) peribronchial growths seen after the endobronchial intrusion of the growth. In the first group the diagnosis can be made in the incipiency by bronchoscopic methods. In several instances the growth was observed so early that there was no recurrence after removal for biopsy. The only reason why an early diagnosis is not more often made is that the patient is not referred to the bronchoscopist at an early stage in the disease.

In malignant growths external to the bronchus there are often found fixity, rigidity and deformity of the bronchial walls in the earlier stages. These are sufficiently characteristic to warrant a tentative diagnosis of malignancy. Histologic confirmation is possible only after bronchial intrusion, which occurs late in the disease. In the third group a positive histologic diagnosis can be made, but this is usually late.

Although first in importance, Jackson advised that diagnostic bronchoscopy should follow, rather than precede, thorough general examination, roentgen study and serologic and other laboratory tests.

In discussing the surgical treatment for bronchial neoplasms, Flick[15] emphasized the importance of early diagnosis. He expressed the belief that progress in the surgical treatment will be made if the diagnostic possibility of bronchial new growth is considered in studying patients with vague pulmonary symptoms. If repeated roentgenograms of the lungs taken over a reasonable time show a suggestive shadow, and if the bronchoscopist cannot see a growth in the bronchus, other conditions being excluded, surgical exploration is indicated. With the present development of thoracic surgery, exploration should be comparatively safe, and if a growth is discovered early, radical removal is feasible. He concluded that bronchoscopy, roentgenology and the use of the thoracoscope and of diagnostic pneumothorax are invaluable as aids in arriving at a diagnosis and in determining the extent of the pulmonary lesion. Future progress lies in the early diagnosis of these growths; then only will they come within the domain of surgery.

In a report of the results secured in surgical treatment in twenty-four cases of intrathoracic new growths, Harrington[16] stressed the impor-

14. Jackson, C.: Malignant Growths of the Lungs: Bronchoscopic Diagnosis, Arch. Otolaryng. **12**:747 (Dec.) 1930.

15. Flick, J. B.: Bronchial Neoplasms, Arch. Otolaryng. **12**:603 (Nov.) 1930.

16. Harrington, S. W.: Intrathoracic New Growths, Surg., Gynec. & Obst. **51**: 647 (Nov.) 1930.

tance of bronchoscopic examination in differential diagnosis, particularly in ruling out primary intrabronchial disease and malignancy. In some cases the presence of an extrinsic tumor may be indicated by narrowing of a bronchus. Differentiation between intrapulmonary and extrapulmonary lesions may often be made by the injection of iodized poppy seed oil, 40 per cent.

Cyst of the Lung.—In Yankauer's [17] patient pulmonary symptoms developed five years after she had been successfully operated on for an echinococcus cyst of the liver. A roentgen study of the chest showed multiple cysts of the right lung, the largest being in the upper lobe. No hooklets or scolices were found in the sputum. At bronchoscopy, performed to secure material for microscopic study, more than half an ounce of slightly milky fluid was secured from a subdivision of the bronchus of the upper lobe of the right lung. The patient was relieved of her symptoms. The cyst, as shown by roentgen study, was about one-half its former size. No hooklets or scolices were found in the fluid; instead, there was observed an ameba-like organism which was identified as belonging to the group of *Trichomonas*. It was believed that this was a secondary parasitic infestation of the original lesion which, in view of the history, must have been hydatid cysts of the lung.

Myerson [18] presented reports of three cases of tuberculosis of the bronchial lymph nodes correctly diagnosed by bronchoscopy. In one case, the bronchial stenosis was believed to be due to a foreign body in a bronchus and enlargement of the thymus gland. In the second case, pneumonia of the lower lobe of the right lung was thought to exist; later, a diagnosis of abscess of the lung was made. At bronchoscopy a macerated tuberculous lymph gland was found in the bronchus. In the third case, one of intractable asthma, lasting five years, bronchoscopy revealed evidences of pressure at the tracheal bifurcation. These cases serve to emphasize the importance of diagnostic bronchoscopy in obscure conditions of the chest and the effect of enlarged tuberculous lymph nodes on the trachea and bronchi.

BRONCHOSCOPY IN TREATMENT

Intrabronchial Drainage.—In a study of cases of pulmonary suppuration, Brunn and Faulkner [19] concluded that the question of drainage

17. Yankauer, S.: Cyst of the Lung: Report of a Case, Arch. Otolaryng. **12**: 649 (Nov.) 1930.

18. Myerson, M. C.: Bronchoscopic Observations of Enlarged Tuberculous Bronchial Glands, Arch. Otolaryng. **12**:627 (Nov.) 1930.

19. Brunn, H., and Faulkner, W. B., Jr.: Intrabronchial Drainage: Its Importance in the Diagnosis and Treatment of Pulmonary Suppuration, Surg., Gynec. & Obst. **51**:115 (July) 1930.

was not fully understood. They found it convenient to designate the spilling of pus or secretion from one bronchus into another on the same or the opposite side as "internal drainage" to distinguish it from "external drainage" or emptying of a bronchus by cough and expectoration. Their studies of the factors controlling intrabronchial drainage are based on bronchoscopic examination and the observation of instilled iodized oil in the tracheobronchial tree. They believed that intrabronchial drainage exerts a considerable influence on the production of symptoms, the course of the disease and its extension both locally and to the opposite side, and that it modifies the physical signs. The symptoms and signs in pulmonary suppuration are often more dependent on the areas to which pus drains than on the actual site of the primary lesion of the lung. The diagnosis and successful treatment of pulmonary suppuration are based on an understanding of intrabronchial drainage.

In a more recent presentation, Faulkner [20] discussed the clinical application of internal drainage and stated that pus within a bronchus may spill to predetermined areas, depending on the posture of the patient, the site of the primary lesion of the lung and the patency of the bronchus. This drainage is especially marked when the pus is of low viscosity and of great quantity. He expressed the belief that a knowledge of the principles of internal drainage has aided in a more accurate localization of pulmonary abscesses, a decrease in the number of patients requiring operation, a lowered operative risk and an increase in the percentage of cures.

Posttonsillectomic Pulmonary Abscess.—Carmody [21] directed attention to the influence that bronchoscopy has exerted in increasing knowledge of pulmonary infections following operative procedures. He discussed the etiology of pulmonary abscess and stated that, although much evidence has been presented to support the embolic theory, the aspiration of material into the air passages during tonsillectomy has been amply demonstrated. One must conclude that no one source of infection can be considered as responsible for all cases. Injury to the tissues may be produced by one organism, and suppuration may be caused by a symbiotic bacterium. The question of atelectasis must be considered. Whether or not infection takes place depends not only on the kind of organism, but on the pathologic changes present. He stated that true abscess is caused by the pneumococcus or by the hemolytic or *viridans* type of streptococcus, while gangrene is caused by the fusospirochete.

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20. Faulkner, W. B., Jr.: Internal Drainage: Its Application in Pulmonary Suppuration, J. A. M. A. **95**:1325 (Nov. 1) 1930.

21. Carmody, T. E.: Pulmonary Abscess Following Tonsillectomy; Laryngologic Aspects, Arch. Otolaryng. **11**:200 (Feb.) 1930.

Pierson [22] emphasized the importance of a carefully secured history and a series of roentgenograms in the diagnosis and management of a case of pulmonary abscess. He believed that the bronchoscopist and surgeon are the most important members and should frequently be in consultation with the internist to aid in deciding on the proper plan of treatment. Some abscesses may be prevented by thorough examination before operation. Anaerobes may be of particular importance in the promotion of abscesses in otherwise merely pneumonic processes.

In the prevention of pulmonary abscess, Pierson suggested the following as possible measures: complete preoperative examination of the patient with special reference to the lungs; hygiene of the mouth before and after operation; the use of local anesthesia whenever possible; the use of a suction apparatus and avoidance of unnecessary crushing of tissue and mass ligatures that favor the formation of thrombi.

In a review of seventy-seven cases of abscess of the lung following tonsillectomy, I [23] found that fifty (65 per cent) occurred in patients between the ages of 20 and 40 years. In forty-two (54.5 per cent) the upper lobe was involved. After discussing the importance of bronchoscopy in the diagnosis and treatment, I concluded that bronchoscopy is indicated as a diagnostic measure in every case of pulmonary abscess in which there is a question of diagnosis. Bronchoscopic aspiration, as a part of the conservative form of treatment, should be instituted early in the disease unless contraindicated; in the hands of an experienced bronchoscopist, contraindications are few. No definite rules can be laid down with regard to the length of time bronchoscopy should be continued; every case of abscess of the lung should be individually considered by the internist, roentgenologist, surgeon and bronchoscopist to determine the form of treatment to be employed.

In discussing the methods by which the bronchoscopist could be of assistance to the thoracic surgeon, Schall [24] stated that, to be of aid, he must appreciate the surgeon's problems and be familiar with the principles of his surgery. The bronchoscopist can improve drainage by the dilation of strictures and by the removal of obstructing granulation tissue. He can give aid to the surgeon in localization of the abscess by tracing the pus stream to its source and by pneumography. In selected cases the bronchoscope can be used as a guide for the surgeon to open an abscess.

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22. Pierson, P. H.: Posttonsillectomic Pulmonary Abscess: Medical Aspects, Arch. Otolaryng. **11**:279 (March) 1930.

23. Clerf, L. H.: Pulmonary Abscess Following Tonsillectomy: Bronchoscopic Considerations, Arch. Otolaryng. **11**:192 (Feb.) 1930.

24. Schall, L. A.: Pulmonary Abscess Following Tonsillectomy: Bronchoscopic Considerations as an Aid to the Surgeon, Arch. Otolaryng. **11**:300 (March) 1930.

Holman [25] restated the generally accepted dictum that the most favorable time for the cure of an abscess is during the period immediately following its inception, and the treatment then will necessarily differ from that required at a later stage when the thickened, rigid walls of the abscess present one of the most baffling problems in surgery. He directed attention to the abscesses resulting from bronchogenic infection. These abscesses are usually due to the many and varied organisms found in the mouth and pharynx and about infected teeth and are of serious import because of the widespread destruction of tissue.

The three important factors in the healing of an abscess cavity are: (1) adequate drainage, (2) the gradual contraction of fibrous tissue laid down as granulations in the wall forming the abscess and (3) the expansion of the healthy normal lung to replace the destroyed lung. In the selection of a plan of treatment it is important to consider these factors. If adequate drainage is impossible through a bronchus, drainage by external operation is indicated.

Funk [26] summed up the forms of treatment for pulmonary abscess under medical, bronchoscopic and surgical. He agreed that bronchoscopy, apart from its value as a diagnostic procedure preliminary to treatment, is of first importance in pulmonary suppuration caused by foreign body. In nonforeign body suppuration, such as posttonsillectomic abscess, it not only aids the overtaxed mechanisms of cough and ciliary action, but frees the air passages from obstructions to the exit of pus. Bronchoscopy renders postural drainage more effective. The former may be done once or twice weekly, or oftener if necessary. He stressed the importance of a bronchoscopic study in all obscure cases of pulmonary abscess, particularly in adults. Several cases of carcinomatous abscess of the lung are cited. New growth was not suspected and, in one, a diagnosis of cancer was made at external operation. At necropsy the bronchial origin of the cancer was demonstrated. In bronchiectasis, bronchoscopy should be done to determine the presence of obstruction, due to foreign body, cicatrices or granulation tissue. In nonobstructive cases, bronchoscopy is of value in aiding the normal forces in getting rid of the secretions and in relieving stagnation.

May and Soulas [27] expressed the belief that bronchoscopy undoubtedly has aided in the treatment of chronic suppurative diseases of the lungs which, heretofore, have been unsuccessfully treated by medical

25. Holman, E.: Posttonsillectomic Pulmonary Abscess: Factors in Healing, Arch. Otolaryng. **11**:287 (March) 1930.

26. Funk, E. H.: The Treatment of Pulmonary Suppuration, Pennsylvania M. J. **33**:725 (Aug.) 1930.

27. May, E., and Soulas, A.: Bronchoscopy in Treatment of Lung Abscess, Bull. et mém. Soc. méd. d. hôp. de Paris **54**:543 (April 7) 1930.

or surgical methods. Three case reports were cited to emphasize certain phases of the general plan of treatment. Emphasis was placed on the value of frequent roentgen examination, also the freedom from danger in bronchoscopic treatment if carried out by one qualified in this work.

Vinson [28] emphasized the fact that discomfort and risks of bronchoscopic procedures are negligible and complications following the operation are rare. Spontaneous pneumothorax may follow bronchoscopic aspiration of a pulmonary abscess and, unless a roentgen examination of the thorax is made after instrumentation, the complication may be overlooked. He reported three cases of pneumothorax occurring after bronchoscopy. In two, the rupture probably occurred in a normal portion of the lung, since effusion did not follow and the lung completely expanded within ten days. In the third case, the rupture probably occurred in an uninfected portion of the lung, as the effusion did not develop until the eleventh day after bronchoscopy.

Bacteriologic Observations in Pulmonary Abscess.—The material for this study was obtained either directly from the abscess cavity at surgical operation or collected by the bronchoscopist. In every case the pus was obtained from the area of infection without contamination by secretions from the mouth. Bucher [29] summarized his observations as follows: Streptococci were most commonly found in these cultures. Staphylococci, pneumococci, *Bacillus influenzae,* fusiform bacilli, spirochetes, various anaerobes and other organisms, some unidentified, were also recovered. It was concluded that the organisms found in pulmonary abscess are similar, for the most part, to those found normally in the mouth and upper respiratory tract. It is not possible, ordinarily, to pick out any one organism as the etiologic factor, but it is believed that many of the organisms found in the abscess cavity gained entrance from the mouth and upper respiratory tract through the air passages.

FOREIGN BODIES IN THE AIR AND FOOD PASSAGES

Bronchial Foreign Bodies.—Sneller [30] cited five cases of bronchial foreign body. He agreed that foreign bodies may enter the tracheobronchial tree without the occurrence of immediate symptoms and emphasized this in the diagnosis; he also stressed the need of ruling out foreign body in every case of suppuration of the lungs and in children with vague symptoms in the chest.

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28. Vinson, P. P.: Spontaneous Pneumothorax Following Bronchoscopic Aspiration of Pulmonary Abscess: Report of Three Cases, M. Clin. North America **13**:1379 (May) 1930.

29. Bucher, C. J.: Abscess of the Lung: A Bacteriologic Study Based on One Hundred and Eighteen Cases, Am. J. M. Sc. **179**:406 (March) 1930.

30. Sneller, C. D.: Important Factors in the Diagnosis of Foreign Bodies in the Air Passages, Illinois M. J. **58**:375 (Nov.) 1930.

The views held by many bronchoscopists concerning the mechanical obstruction produced by foreign bodies in the air passages, namely, obstructive emphysema and obstructive atelectasis, are not generally shared. In the estimation of certain observers, the obstruction that is responsible for the phenomena of emphysema and atelectasis is produced by spasm of the bronchial musculature, probably being induced by the presence of the foreign body.

Aucoin [31] reported seven cases of bronchial foreign bodies, giving full clinical and roentgen details. His contention was that the changes produced are the result of mechanical valve action. He concluded that bilateral pulmonary emphysema may result from obstruction of the larynx or trachea, particularly when marked expiratory dyspnea is present. If a condition of spasm of the bronchial musculature does exist, he has never observed it during bronchoscopy. Partial obstruction of a bronchus is accompanied by emphysema of the lung beyond the obstructed area, while complete blocking of a bronchus is accompanied by a collapse of the lung beyond the obstruction.

Arachidic Bronchitis.—In an experimental study, Heatly and Clausen [32] were able to produce typical arachidic bronchitis in the rabbit. They demonstrated that arachidic bronchitis was due to irritation, whereas infection played only a secondary rôle. The fat-free residue of peanuts containing unaltered proteins and carbohydrates produced no reaction. The irritant found is an acid present in both the oleic and the linolic fraction and absent in the solid fraction of the fatty acids. Peanut oil containing free fatty acids is irritating, whereas peanut oil deprived of free fatty acids is nonirritating. The concentration of free acids that confers irritating properties on peanut oil may be extremely small, e. g., 0.3 per cent.

Gittins [33] reported the case of an infant, aged 22 months, in whose bronchus and trachea a peanut was present for sixty-three days before a diagnosis was made. Although there was a definite history of choking while eating crackerjack, and the child had been observed during three attacks of pneumonia (?), foreign body was not suspected, nor did any of the attending physicians advise bronchoscopy. Gittins found little secretion and slight reaction in the mucosa of the trachea and bronchi; he attributed this to the fact that there was only one foreign body, and this was freely movable in the trachea and bronchus.

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31. Aucoin, E. L.: Foreign Bodies in the Air Passages: Emphysema and Atelectasis by Obstruction, Arch. internat. de laryng. **9**:622 (June) 1930.

32. Heatly, C. A., and Clausen, S. W.: Experimental Studies in Peanut Bronchitis, Arch. Otolaryng. **11**:569 (May) 1930.

33. Gittins, T. R.: Peanut in the Bronchus of an Infant Aged Twenty-Two Months: Bronchoscopy with Recovery, Arch. Otolaryng. **11**:614 (May) 1930.

Negus [34] reported three cases of nonopaque foreign body in the right bronchus. In one case, in which a rabbit's vertebra had been aspirated sixteen months before, there was marked pulmonary suppuration. The removal of the foreign body was subsequently followed by complete recovery. In the remaining two cases, the roentgen observations were characteristic of obstructive emphysema of a marked degree. Following removal of the obstructing foreign bodies, the chest promptly became normal. Negus emphasized the need for the safe and harmless procedure of bronchoscopy in unexplained cases of pulmonary disease and suggested to the throat surgeon that it would appear that under certain conditions the bronchoscope is at least as important an instrument of examination as the stethoscope.

In a statistical review, Jackson [35] found that bones constituted about 15 per cent of foreign bodies and that over 90 per cent of these were in the food passages. He also observed that bones, occurring as foreign bodies in the lungs, were more often "overlooked" than other foreign bodies. Brief reports of ten cases of overlooked bones in the air passages are quoted.

In answer to the query why the bones often remained undiagnosed for such long periods, Jackson pointed out three of the most important factors, namely: a symptomless interval followed the accidents; physical signs, though present, were capable of being otherwise interpreted, and the roentgenograms showed a pathologic process; however, in all but two cases the films were negative for a shadow of a foreign body. A history of foreign body, though present in all but two cases, either was not elicited, or was not given sufficient attention. He expressed the belief that foreign body must always be thought of in the diagnosis of chronic pulmonary disease, whether or not there is a suggestive history, whether or not physical signs are pathognomonic, whether or not the roentgenogram shows a suggestive shadow, and even if the first bronchoscopy proves negative.

In Hara's [36] case there was a definite history of initial symptoms at the time of the accident together with the development of symptoms and signs referable to the chest. Cough, blood-streaked expectoration, fever and slight dyspnea persisted and were relieved by bronchoscopic removal of a large foreign body one month after the accident.

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34. Negus, V. E.: Three Cases of Nonopaque Foreign Body in the Right Bronchus, J. Laryng. & Otol. **45**:119 (Feb.) 1930.

35. Jackson, C. L.: Bones as Overlooked Foreign Bodies in the Lung, Arch. Otolaryng. **12**:499 (Oct.) 1930.

36. Hara, H. J.: Large Pork Bone in Bronchus, California & West. Med. **33**:606 (Aug.) 1930.

Miller [37] reported a case of obstruction of the left main bronchus by the electric bulb of an otoscope. When first seen by the family physician, the outstanding symptoms complained of were epigastric pain and vomiting. There was a history of injury to the chest; however, the only possible evidences of a traumatic lesion were subcutaneous emphysema over the left anterior part of the chest and physical signs of a partially localized left-sided pneumothorax. By roentgen examination an opaque foreign body was found in the left bronchus. Although there was a marked endobronchial inflammatory reaction, the foreign body was successfully removed and the patient made a complete recovery.

Jesberg [38] analyzed a series of 213 foreign bodies removed from the air and food passages and found that disks were the most common form of foreign body (22 per cent). He agreed that disks rarely enter the respiratory tract and that the usual site of lodgment in the esophagus is below the level of the cricopharyngeus muscle at the superior thoracic aperture. This he explained on anatomic grounds, together with the fact that the surface area of that part of the disk in contact with the lateral walls of the esophagus is much smaller than that of an ordinary bolus of food.

The esophagoscopic removal of a Star safety razor blade, measuring 40 by 20 mm., from the lower end of the thoracic esophagus was reported by Imperatori.[39] A portion of the blade was found to have passed through the hiatus into the cardia. By careful protection and tilting of the edge of the blade it was removed without producing any trauma, with the exception of slight bleeding from the cardia. The patient made an uneventful recovery.

Orton [40] presented six cases of mediastinitis complicating foreign bodies in the esophagus. Because of the serious nature of this condition and the difficulties encountered in the surgical treatment there was considerable question as to the proper method of procedure. In two of the six patients esophagoscopy was performed in the presence of mediastinitis and both of the patients died. In two, with mediastinitis, no endoscopic procedure was carried out and both patients died. In one case in which the foreign body consisted of a safety-pin, the point of which had perforated the lower end of the esophagus, a gastrostomy was performed and the pin removed from below. The patient died

37. Miller, J. W.: Endobronchial Foreign Bodies, Arch. Otolaryng. **11**:772 (June) 1930.

38. Jesberg, S.: Disks in the Esophagus, Arch. Otolaryng. **11**:210 (Feb.) 1930.

39. Imperatori, C. J.: An Unusual Foreign Body, Arch. Otolaryng. **11**:213 (Feb.) 1930.

40. Orton, H. B.: Mediastinitis Following Esophageal Foreign Body: Report of Cases, Arch. Otolaryng. **12**:635 (Nov.) 1930.

twenty-two days later of suppurative mediastinitis. In the remaining case, no esophagoscopy was done by Orton until nature had attempted to wall off the pus so that external drainage could be carried out. A coin was then removed from the esophagus, and the patient ultimately recovered. Orton expressed the belief that endoscopic procedures should be delayed until the infected area had been walled off, and that the esophagus should be placed at rest by gastrostomy.

DIRECT LARYNGOSCOPY

Direct Laryngoscopy in Laryngeal Obstruction.—Richards [41] directed attention to the often overlooked fact that not every case of acute obstruction of the larynx could be handled in the same manner; if, therefore, time permitting, an accurate diagnosis could be made and the exact nature of the condition could be determined, the ultimate outcome would be more satisfactory. In Richards' experience there was no one method of procedure which allowed one more carefully to size up the situation, to make a more accurate diagnosis, or, in some instances, to carry out the logical form of treatment as direct laryngoscopy. It enabled one, first of all, to locate the point of obstruction with relation to the laryngeal structures. It disclosed the existence of an unsuspected foreign body, membrane or subglottic swelling. Frequently conditions were found which could either be instrumentally removed or temporarily relieved. He arrived at the conclusion that every case of acute laryngeal obstruction should sooner or later be examined by direct vision.

DISEASES OF THE ESOPHAGUS

Congenital Anomaly of the Esophagus.—Phelps [42] was convinced that congenital anomalies of the esophagus are not rare. He described nine definite varieties of anomalies and explained the occurrence of certain of these on an embryologic or evolutionary basis; in others, no adequate explanation could be given. In some of these cases the patients can be treated successfully. Phelps stressed the importance of an accurate diagnosis in every case. In certain of his cases it was necessary to perform an esophagoscopy in order to establish a correct diagnosis.

Kelly [43] reported four cases of congenital esophageal stenosis in young children. There was found an associated hernia of the abdominal

41. Richards, L. G.: Acute Laryngeal Obstruction, J. A. M. A. **95**:766 (Sept. 13) 1930.

42. Phelps, K. A.: Congenital Anomalies of the Esophagus with a Report of Nine Cases, Ann. Otol. Rhin. & Laryng. **39**:364 (June) 1930.

43. Kelly, A. B.: Congenital Stenosis of the Esophagus in Children, Associated with Diaphragmatic Hernia of the Stomach, J. Laryng. & Otol. **45**:680 (Oct.) 1930.

esophagus and part of the stomach, through the hiatus esophageus, into the thoracic cavity. In all of the cases, persistent vomiting or regurgitation was the outstanding symptom. This had set in immediately or a few days after birth. The roentgenograms were difficult to interpret, but sufficient additional data were secured by esophagoscopy to aid in making a diagnosis. Kelly was not convinced that the association of the congenital esophageal stenosis and the hernia was accidental. The conditions are probably examples of multiple congenital abnormalities. He concluded that many of these cases have probably been overlooked because of the difficulty of demonstrating the hernia by roentgen examination. To accomplish this, it is necessary to distend the sac with a barium mixture.

Angioneurotic Edema, Urticaria, Serum Disease and Herpes of the Esophagus.—In presenting a report of cases of angioneurotic edema, urticaria, serum disease and herpes of the esophagus, Jackson [44] stated that, although additional knowledge of these conditions might require a rearrangement of the names and classification, carefully made clinical observations recorded accurately and separated from inferences would always remain clinical facts. He concluded that angioneurotic edema, urticaria, serum disease and herpes could involve the esophageal mucosa. Herpes is distinguished by blisters or blebs that soon break, leaving a raw, eroded surface, which is later covered with exudate. Angioneurotic edema, urticaria and serum disease of the esophagus are associated with other similar manifestations elsewhere. Their affinities may preclude differential diagnosis as among them and they may be essentially the same; however, there is no difficulty in distinguishing them from other lesions of the esophagus, provided the esophagoscope is used. The esophagoscope is the only means by which the diagnosis can be made with certainty.

Farrell [45] emphasized the importance of a careful roentgen study of the function of swallowing of every patient complaining of the slightest disturbance in swallowing. A detailed technic was given, and the differential diagnoses of the various pathologic conditions producing dysphagia were enumerated. Although roentgenology does not afford the accuracy in diagnosis that can be obtained by esophagoscopy, it is vastly superior to blind bougienage. Farrell condemned bougienage as being dangerous, usually uninformative and never entirely conclusive. He concluded that esophagoscopy should be employed in all conditions

44. Jackson, C.: Diseases of the Esophagus: Angioneurotic Edema, Urticaria, Serum Disease and Herpes, Arch. Otolaryng. **11**:397 (April) 1930.

45. Farrell, J. T., Jr.: Roentgen Diagnosis of Cancer of the Esophagus, Radiology **14**:282 (March) 1930.

in which a positive opinion as to the presence or absence of organic disease of the esophagus cannot be given by roentgen study.

Esophageal Changes Due to Cardiac and Aortic Diseases.—The importance of changes in the esophagus secondary to diseases of the heart and aorta is of interest to esophagoscopists. Rigler and Phelps [46] stated that most of these patients present no esophageal symptoms, and esophagoscopy generally is contraindicated; they expressed the opinion, however, that certain phases of the subject are of some practical value. They found that dilatation of the aortic arch produced a deviation of the esophagus to the right, posteriorly with compression. Dilatation of the descending aorta displaced the esophagus anteriorly and produced compression; if in the proximal three-fourths, the esophagus was displaced to the right, while in the distal fourth it might be displaced to the left. Dilatation of the left auricle compressed the esophagus and displaced it posteriorly.

46. Rigler, L., and Phelps, K. A.: Changes in the Esophagus Secondary to Cardiac and Aortic Diseases, Arch. Otolaryng. **11**:188 (Feb.) 1930.

Abstracts from Current Literature

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HEREDITARY DEAFNESS DUE TO BILATERAL ACOUSTIC TUMORS. W. JAMES GARDNER and CHARLES H. FRAZIER, Ann. Otol., Rhin. & Laryng. **39**:974 (Dec.) 1930.

Hereditary deafness resulting from bilateral acoustic tumors has not hitherto been recorded, according to the authors, who report the results of an investigation suggested by a statement in the history of such a patient which intimated deafness in a number of the family connections for five generations. The survey of this family disclosed a history of bilateral deafness in thirty-eight members, with subsequent blindness in fifteen instances. Of the deaf and blind persons, four were examined prior to death and were found to have choking of the optic disks with secondary atrophy. The seven affected members living were personally examined and presented clinical indications of bilateral acoustic tumors. The presence of these lesions was proved by necropsy in two cases.

The patient whose history stimulated the investigation was a man, aged 28, whose illness was essentially a repetition of a common complaint that had affected many members of his family for five generations. He had noted a defect in his hearing at the age of 17. This increased steadily and became complete six weeks prior to his admission to the hospital, coincident with a beginning failure of vision. The defect in vision progressed rapidly, total blindness resulting two weeks before admission. After the development of complete deafness there was nausea and vomiting, while vertigo and a staggering gait had been present for four months. Whether or not tinnitus was a symptom could not be determined. In order to communicate with the patient it was necessary to spell out the words on his right palm, using his left index finger to draw the letters. This method was used by all members of this family who had become deaf and blind. Examination revealed bilateral choked disks with numerous hemorrhages, resulting in total blindness. There was complete loss of the sense of smell and complete loss of function of both eighth nerves in both the cochlear and vestibular portions. A diagnosis of bilateral acoustic tumors was made, and suboccipital craniotomy was performed. A large acoustic tumor was found on the right side and partially removed by the intracapsular method. No tumor was found on the left side. The patient died three days later from a hemorrhage of unknown origin into the ventricles, and necropsy disclosed an acoustic tumor on the left side which was smaller than that on the right side. There were no other tumors of the brain or meninges.

Histologic examination revealed the lesions to be not of the nature of the ordinary isolated acoustic tumor, or perineural fibroblastoma, in which the nerve fibers are present only in the capsule, but of the nature of the true neurofibroma of Recklinghausen's disease, in which the nerve fibers are found within the tumor tissue proper. The histologic diagnosis was bilateral acoustic neurofibromas. In this family the condition was transmitted as a true mendelian dominant character, as may be readily seen by the charts that accompany the authors' report.

SNAPP, Grand Rapids, Mich.

THE SUPPURATION OF THE PETROUS PYRAMID: PATHOLOGY, SYMPTOMATOLOGY AND SURGICAL TREATMENT. SAMUEL J. KOPETZKY and RALPH ALMOUR, Ann. Otol., Rhin. & Laryng. **39**:996 (Dec.) 1930.

In the course of extended study of otitic meningitis, it became apparent to the authors that, in the interval of time which elapses in most cases between the otitic and mastoid lesion and the therapy applicable to them, on the one hand,

and the terminal lesion in the meninges, on the other, there ensues a period in which the patient is almost free from symptoms. The importance of this interval has been demonstrated by establishing a relationship between the original infection and the terminal meningitis. This was accomplished by the aid of roentgenograms of the petrosal pyramid during life and before the onset of the terminal meningitis, thus making the diagnosis of the lesion in the petrous pyramid more exact and establishing it as a distinct clinical entity. Considerable confusion exists regarding the so-called Gradenigo syndrome, which is the term given to the pathologic changes underlying those cases which present pain in the area of the trifacial nerve and paralysis of the abducens nerve, sometimes accompanied, but more often unaccompanied, by changes in the cell content of the cerebrospinal fluid. The authors state that they are not dealing with this syndrome except incidentally; they, along with other workers, hold that the series of events that are usually characterized by the term Gradenigo syndrome are not characteristic of a specific lesion and do not form a distinct clinical entity. Gradenigo, in his original studies, brought forward no histologic evidence to support his belief that the triad of symptoms that he described is diagnostic of any one organic pathologic condition. Various workers disagree with Gradenigo, stating that the triad does not furnish the typical picture of the disease, and also that paralysis of the abducens nerve appears only in the winter months, is accompanied by other evidences of grip and hence is a toxic neuritis. Others claim that it is reflex in nature. Alt gave six etiologic factors for the causation of the syndrome, while Lange stated that when widespread abscesses in the immediate vicinity of the abducens nerve cause no paralysis, one cannot take the stand that there is more than a casual relationship. The authors wish to reiterate their belief in the Wittmaack theory of pneumatization of the mastoid process, supported as it is by histologic, roentgenographic and clinical observations. It is on this work that they base their explanation of the mechanics of invasion of the petrosal pyramid, of the symptomatology following invasion of the perilabyrinthine air cells and of the causation of chronicity in infections of the middle ear. Suppurations of the petrosal pyramid are of two varieties: frank suppurations of the pyramid, more particularly its tip, and osteomyelitis of the pyramid. The latter is of rare occurrence and is present in diploetic rather than pneumatized bone. It is not discussed in this report.

In a well pneumatized temporal bone the pneumatic cells are by no means limited to the mastoid portion of the bone, but also occupy the squamous and zygomatic portions. They are found in the floor of the middle ear, under the tegmen and more extensively around the mouth of the eustachian tube. Keeping in mind the Wittmaack theory of pneumatization, it thus becomes evident that wherever there are marrow spaces in contact with tympanic mucosa the latter can exert its pneumatizing influence and convert them into air-containing chambers, which will then communicate directly with the middle ear. Consequently, the entire petrous pyramid may be filled with large pneumatic spaces, being especially well developed in the petrosal tip and in the perilabyrinthine area. Other anatomic factors of importance in the comprehension of the lesion are the relationships of the various soft tissue structures which come in contact with or pass through the temporal bone, namely, the membranous labyrinth, the fifth, sixth, seventh, eighth, ninth, tenth and eleventh nerves, the carotid artery, the eustachian tube and the petrosal nerves.

In suppuration of the petrosal tip, the avenues of infection are: from the antrum or epitympanic space, above or below the superior semicircular canal, following the posterosuperior surface of the petrosal into the pyramidal tip; from the peritubal cells into the pyramidal tip; and from the peritubal cells directly into the carotid canal or through dehiscences in the anterior tympanic wall into the carotid canal and then rupturing into the cavum mecklii. From a clinical observation of nine patients with suppuration of the petrosal tip, five of whom recovered, the authors add to the postmortem observations several additional factors which can be observed during the progress of the case through the various stages. All of their cases showed extensively pneumatized mastoid processes. As the suppu-

ration in the mastoid process and tympanum clears up, the process spreads into the perilabyrinthine tracts toward the pyramid. After a period during which the middle ear remains dry, there suddenly reappears a profuse aural discharge, as a source of which the mastoid wound can be definitely ruled out, for it appears healthy and contains no pus. It is concluded that the pus within the pyramid finds an avenue of escape by rupturing through the cells around the eustachian tube and then out through the middle ear. With a protracted acute infection of the middle ear after operation, one must think of suppuration of the pyramidal tip.

SNAPP, Grand Rapids, Mich.

RELATION OF INFECTION OF THE EAR AND INFECTION OF THE INTESTINAL TRACT IN INFANTS. D. E. S. WISHART, J. A. M. A. **95**:1084 (Oct. 11) 1930.

This comprehensive study attempts to answer the question: Is infection of the mastoid the cause of acute intestinal intoxication in infants? The author's answer is: No. The onset of the disease was rarely characterized by a "cold"; the great majority of the infants were free from clinical evidence of infection of the upper respiratory tract during the toxic state; many were free from aural infection throughout the whole course of the illness, and accumulations or infections in the mastoid antrums at autopsy were of antemortem origin. Both ear drums of many infants were abnormal immediately preceding death, but this change was the result of the antemortem ejection of vomitus into the eustachian tubes. Mastoid infection was the result and not the cause of the child's lowered condition. Puncture of the mastoid antrum for diagnosis should not be recommended and mastoidectomy as a cure was a failure. Operation should be postponed as long as possible. There was no correspondence in bacteriology between the infection in the upper respiratory tract and that in the intestinal tract. Accumulating evidence indicates that the disease is of intestinal origin.

BONAR, Salt Lake City.

THE DEVELOPMENT OF THE EAR OF ACANTHIAS VULGARIS. D. P. QUIRING, J. Morphol. & Physiol. **50**:259 (Sept. 5) 1930.

The first indications of auditory vesicles in *Acanthias vulgaris* occur in embryos of from 3.5 to 4 mm. in length, consisting of thickened ectodermal areas composed of large columnar cells. No indication of the invagination of the placodes is present in this stage. In embryos of from 8 to 12 mm. in length, increase in size without differentiation has taken place. In embryos from 15 to 20 mm., differentiation has set in; this is most apparent in the bulging of the walls of the vesicles, indicating the position of future canals. The endolymphatic duct is present. The vesicle is attached to the ganglion of the auditory nerve. A well differentiated sacculus and utriculus, as well as the lagena, are first noted in the 22 mm. stage. Complete separation of the canals, the utriculus, the sacculus, the lagena and the recessus utriculus has taken place in the 30 to 38 mm. stage, together with definite innervation of the ampullae and other sensory areas. The three ampullae are lined with ciliated sensory cells as well as the recessus utriculus, macula neglecta, sacculus and lagena. The utriculus has no sensory patch. The adult structure has three canals, three ampullae, a sacculus, utriculus, lagena, recessus utriculus and rami of the eighth nerve supplying the separate parts. The endolymphatic duct remains open to the surface in the adult form.

WYMAN, Boston.

SENSE OF HEARING SURVEY OF SCHOOL CHILDREN IN FERGUS FALLS. W. L. BURNAP, Minnesota Med. **12**:691 (Nov.) 1929.

Burnap's conclusions are based on the examination of 1,525 school children. Over a third of the children with poor hearing have disease processes present, the removal of which should wholly or partially remedy the defect, while others have permanent injury which could have been prevented. About 8 per cent of

the school children of Minnesota have defects in hearing greater than 6 sensation units. The audiometer is a rapid, accurate and convenient instrument for determining acuity of hearing.

HUENEKENS, Minneapolis.

MIDDLE EAR INFECTION: COMMON FORMS AND THEIR MANAGEMENT. JOSEPH POPPER, New York J. Med. **30**:1146 (Oct. 1) 1930.

The following indications are given for myringotomy: (1) an inflamed ear drum plus severe pain which is not relieved by palliative measures, (2) an inflamed ear drum plus high fever not otherwise accounted for and (3) an inflamed drum plus marked tenderness in the mastoid area. In most cases the drum is bulging if these signs are present. In some infants with mastoid tenderness, relief may be obtained by an incision in the superoposterior wall, thus preventing operative mastoiditis. "Where the clinical picture does not warrant it, positive x-ray findings should not influence the otologist in favor of operation."

Insufflation of iodine powder (Sulzberger) is recommended to clear up the discharge in chronic otitis media.

AIKMAN, Rochester, N. Y.

ETIOLOGY OF MIDDLE EAR SUPPURATION. M. M. CULLOM, South. M. J. **23**: 1091 (Dec.) 1930.

The author believes that purulent disease of the accessory sinuses is by far the greatest cause of suppuration of the middle ear. He is also convinced that purulent infection of the middle ear is by direct continuity and is not blood borne. Purulent infection of the accessory sinuses is commonly overlooked. Cullom also states that some abscesses of the lung are the result of purulent infection of the sinuses.

SCHLUTZ, Chicago.

CLINICAL IMPORTANCE OF STRUCTURE OF PYRAMIDAL APEX. BELINOFF and M. BALAN, Monatschr. f. Ohrenh. **64**:1185 (Oct.) 1930.

Belinoff and Balan stress that a purulent process may sometimes spread from the tympanic cavity or from the mastoid antrum to the cells of the pyramidal apex without, or slightly, involving the pneumatic spaces of the mastoid process. A case of suppurative otitis media was observed in which the mastoid process appeared free from pus on antrotomy, while a purulent infiltration was found in the cells of the pars petrosa. These cases, which are considered as petritis or petro-apicitis, depend on the anatomic structure of the pyramid of the tympanum. The authors studied the pyramid of forty temporal bones removed from middle-aged persons, and they distinguish between three types of pyramidal apex, namely, the diploic, the pneumatic and the mixed types. The diploic type, or deficient pneumatization, was encountered in 22.5 per cent; the pneumatic, or perfect type, in 35 per cent and the mixed, or spongiosodiploic type, in 42.5 per cent of the cases. In cases of otitis media, the menace of transmission of the purulent agent to the pyramidal apex is great when the pneumatic spaces of the pyramid are well developed and the mastoid process is compact. In 37.5 per cent of the cases, the structure of the mastoid process and that of the pyramidal apex was identical; in 62.5 per cent, their structure differed. Coexistence of a well pneumatized pyramidal apex with a diploic or spongiopneumatic mastoid process favors the spreading of suppuration from the mastoid antrum to the pneumatic spaces of the pyramidal apex. The authors add that the means of communication of the antrum with the pyramid will be treated in another article

EDITOR'S ABSTRACT.

CASE OF ATAXIC APHASIA WITH PARALYSIS OF ARM FOLLOWING OTITIC SINUS THROMBOSIS. MANN, Monatschr. f. Ohrenh. **64**:1207 (Oct.) 1930.

Mann's case occurred in a man, aged 21. Eight years before, the patient had had pneumonia and suppurative otitis media. Fourteen days prior to his admission

to the hospital, a purulent discharge reappeared in the left ear; it was followed by severe chills, headache and fever. On examination, the right ear proved to be normal; on the left side, a large defect of the tympanic membrane, traces of pus and tenderness of the mastoid process were found. The patient's history and the objective signs suggested the diagnosis of sinus thrombus. On the basis of tenderness at the anterior border of the sternocleidomastoid muscle, the presence of chills and the poor general condition, it was presumed that was suppuration of the thrombus and spread of the process to the internal jugular vein. The first stage of the operation consisted in ligation of the jugular vein at the level of the thyroid cartilage, whereas the tread of the distal end was left outside. The vein was not slit on account of a menacing hemorrhage. The second stage of the operation consisted in chiseling of the temporal bone and in exposure of the sigmoid sinus. Two cells filled with pus were located close to the lower curve of the sinus; a solid thrombus was found therein. The thrombus was removed and tampons were inserted. Improvement resulted lasting for ten days. Then as fever, severe toothache in the left side of the upper jaw, headache and mental disturbances developed, a radical operation was performed. A few granulations were present in the antrum and in the tympanic cavity; pus was absent. On the third day after the operation, ataxic aphasia and paralysis of the right arm developed, testifying to the presence of a focus in Broca's area and in the arm center. The involved region was exposed. No pus escaped on puncture of the dura; the cerebral mass was covered with a fibrinous tissue, especially along the vessels. The brain had a gelatinous aspect; puncture of the Broca's area and of the arm center was negative. The speech gradually improved, and the paralysis vanished. The patient was discharged with a small fistula at the place of the trephination; the wound from the radical operation healed completely and the general condition was good. The patient was repeatedly examined during the last three years; the condition continued to be good. Mann presumes that a congenital stenosis of the sinus and of the jugular vein on the intact side was responsible for the chronic venous stasis. Ligation of the jugular vein brought about suppuration of the distal portion of the vein. The author thinks that the severe complications could be prevented if this portion of the vein would be slit the next day after the ligation. The severe headache is explained by an increase of the stasis, and the toothache by compression of the gasserian ganglion from the distended cavernous sinus. Moreover, the ligation might have resulted in exclusion of the inferior petrosal sinus which aggravated the stasis in the cavernous sinus. Further, deficient functioning of the jugular vein caused venous stasis in the frontal portion of the brain, and consequently aphasia and paralysis. The author adds that such severe complications from ligation of the jugular vein are rare.

EDITOR'S ABSTRACT.

USE OF PARAFFIN OIL FOR DRESSING OF WOUNDS AFTER OPERATIONS ON THE MASTOID. H. FREY, Monatschr. f. Ohrenh. **64**:1215 (Oct.) 1930.

Frey advocates the method of moistening strips or wicks of iodoform gauze with paraffin or petrolatum oil, in otologic practice. The purpose of the oil is to relieve the discomfort and even pain accompanying the removal of dressing after an operation on the mastoid process. The wicks are inserted through the auditory canal or the retro-auricular wound. The tamponade should not be compact, therefore the flaps are fixed with one or two catgut sutures. The author points out that owing to the hemostatic action of petrolatum, the bleeding from the surgical wound is reduced. No blood clots or crusts irritate the surface of the wound. The wicks or strips must be thoroughly moistened in order to prevent adherence of the external dressing and to allow free passage of the secretory discharge. Frey prefers the method to introduction of glass tubes or rubber drains.

EDITOR'S ABSTRACT.

OCULOMOTOR REFLEXES PROVOKED BY SOUNDS IN PERSONS WITH DIFFICULT HEARING. E. FRÖSCHELS, Ztschr. f. Hals-, Nasen- u. Ohrenh. **26**:511 (Nov. 1) 1930.

Fröschels' method for ascertaining whether there is at least a slight perception of sound was as follows: The sounds from Urbantschitsch's harmonica were directed by means of an otoscope to the auditory canal of persons who were hard of hearing. This was frequently followed by swinging of the patient's body or by rotation of his eyeballs, and sometimes by both. Insufflation of the patient's ears merely with air failed to provoke such an oculomotor reflex. The eyeball moved first to the side opposite the irritated ear, then back to its initial position. The direction of the movements was often horizontal; sometimes rotatory. In a few cases the movements were directed upward or vertically. The movements varied with changes in the sounds. In cases in which hearing was different in both ears, the direction of the movements was from the side of the less affected ear. The direction was not affected by changes in the position of the head. Sometimes the oculomotor reflex appeared when the musical instrument was placed at a distance of 3 or 4 cm. from the ear (without an otoscope); evidently the cochlear nerve is involved in the reflex. The reflex failed when the tests were made with a tuning fork without or with an otoscope. Once the test was applied to a patient during an induced rotatory nystagmus. The nystagmus did not prevent the reaction of the eyeball in response to sounds of a whistle. The reflex became less intense or even disappeared after the test has been frequently repeated. The author queries whether a twitching of the eyeballs, possibly also of the head and of the body, were not habitual in the primitive man; thus the reflex is partially explained by atavism.

EDITOR'S ABSTRACT.

HISTOLOGIC STUDY OF FORMATION OF CHOLESTEATOMA. W. DÖDERLEIN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **26**:521 (Nov. 1) 1930.

The formation of cholesteatoma in the middle ear is explained as follows: An epidermal metaplasia develops in the course of chronic otitis media. Most frequently the epidermis penetrates through a perforation in the tympanic membrane; more rarely, through a fistula in the bone of the auditory meatus. The epidermis covers erosions located around the perforation, but does not invade the epithelium; the epidermis and the epithelium become directly joined, whereby junction is characterized by a thickening. The epidermis may also invade through an infolding of the tympanic membrane at the region of Shrapnell's membrane, induced by the difference of air pressure in the middle ear and in the auditory meatus. In cases in which ersions are absent, the epidermis penetrates into the subepithelial tissue, under the form of cones. This is the most important occurrence connected with a pathologic condition of the submucosa characterized by an inflammatory infiltration of the submucous connective tissue or by the presence of granulation tissue. Existence of a free pus particularly favors the penetration of epidermis, which ceases when the granulation tissue starts to regress, and is replaced by a connective tissue. It is followed by resorption of the penetrating epidermis and splitting of the epidermal cones into clumps. Cells located in the center of the epidermal clumps become disintegrated, thus forming a greasy paplike mass rich in cholesterol. The cell clumps are surrounded by a ring of connective tissue containing elastic fibers. Döderlein considers that the surrounding connective tissue assimilates with the preserved epidermal clumps. Hence a new tissue or tumor develops, composed of epidermis and of connective tissue. The morphologic and histologic features of the tumor correspond to those of a cholesteatoma.

EDITOR'S ABSTRACT.

ROENTGENOGRAPHIC STUDY OF PNEUMATIZATION OF TEMPORAL BONE IN OTOSCLEROSIS. J. G. DILLON and B. S. PREOBRASCHENSKY, Ztschr. f. Hals-, Nasen- u. Ohrenh. **26**:537 (Nov. 1) 1930.

Sonnenkalb's method was used by Dillon and Preobraschensky for study of 100 temporal bones belonging to 50 patients with otosclerosis. The age of the

patients varied from 16 to 54. On the basis of the observations, the cases were classified in three groups. The first group included 20 cases in which the pneumatization was pronounced; in all of them the pneumatization of the mastoid process was extensive. In 13, the squamous part as well as the base of the zygomatic process were involved and in 6, the pyramid. The second group included 4 cases in which 1 temporal bone was of the pneumatic type and the other temporal bone of the scleropneumatic type; 11 cases in which 1 temporal bone was of the sclerotic type, and the other of the scleropneumatic type; and 4 cases in which both temporal bones were of the mixed type. In the third group were included 11 cases with considerable sclerosis of the temporal bone. In some cases small air cells were situated near the antrum; in 1 case, such cells were found around the right sigmoid sinus; in 3 cases, the cells were present in the squamous part and in the zygomatic process; in 1 case, a terminal cell was located at the apex of each of the two mastoid processes and in another, a few cells were found along the left lambdoid suture. The pneumatization of the temporal bone was not characteristic in patients with otosclerosis, and the structure of the temporal bone did not appear to have any appreciable influence on the course of the otosclerotic process. Moreover, the results indicated that the otosclerotic process could not affect the structure of the bone, since the pneumatization of the temporal bone was complete before the otosclerotic process developed. The disturbances of the endocrine glands were not pronounced enough to cause changes in the temporal bone.

EDITOR'S ABSTRACT.

Pharynx

RESULTS OF TONSILLECTOMY. ALBERT D. KAISER, J. A. M. A. **95**:837 (Sept. 20) 1930.

In spite of the favorable evidence regarding the relationship of infected tonsils to disease, there is some doubt of its value in the great mass of children subjected to tonsillectomy. In urban communities about 50 per cent of the older children have had their tonsils and adenoids removed. The author asks the question, "Is this procedure wise and beneficial to the child?" A comparison of 1,000 children of high school age who had been operated on was made with a similar number in whom the operation had been advised but not performed. A similar survey was made of 2,400 children three years after the removal of the tonsils. Kaiser concludes that the real value of removal of tonsils and adenoids cannot be established in a few years. There are substantial benefits in influencing the incidence of sore throat and rendering children less susceptible to diphtheria and scarlet fever. Acute colds in the head and otitis media are not essentially influenced over a ten year period. Cervical adenitis is decidedly reduced. Laryngitis, bronchitis and pneumonia are not lessened, but actually occur more frequently in tonsillectomized children. First attacks of rheumatic manifestations occur from 30 to 50 per cent less frequently in tonsillectomized children, but no effect is produced on recurrent attacks. Incomplete tonsillectomies do not offer the same protection against the usual infections of the throat as complete operations.

BONAR, Salt Lake City.

EMPHYSEMA OF THE HEAD AND NECK COMPLICATING TONSILLECTOMY. F. H. VON HOFE, J. A. M. A. **95**:934 (Sept. 27) 1930.

Three cases of emphysema of the head and neck following tonsillectomy are presented. It is probable that air may enter following perforation of the bed of the tonsillar fossae, by perforation of alveoli of the lung or by being forced into Wharton's duct and thence diffused.

BONAR, Salt Lake City.

TONSILLECTOMY IN THE TREATMENT OF ACUTE CERVICAL ADENITIS IN CHILDREN. HARRY L. BAUM, J. A. M. A. **95**:1829 (Dec. 13) 1930.

The author defends the principle that it is safe to remove tonsils and adenoids during the height of an acute anterior cervical adenitis, and he advocates this procedure.

BONAR, Salt Lake City.

SPORADIC SEPTIC SORE THROAT DUE TO STREPTOCOCCUS EPIDEMICUS. ISADORE PILOT and DAVID J. DAVIS, J. Infect. Dis. **47**:507 (Dec.) 1930.

Three cases of sporadic tonsillitis and sore throat of undetermined origin were described. It was suggested that carriers of *Streptococcus epidemicus* were the source of infection, although milk sources could not be definitely ruled out. *Streptococcus epidemicus* may be the cause of infections of the middle ear and mastoid associated with mild inflammation of the throat and upper respiratory passages without acute tonsillitis.

TOOMEY, Cleveland.

TONSILLECTOMY BY THE BALLENGER-SLUDER METHOD, THE OPERATION OF CHOICE IN CHILDREN. G. CANUYT, Presse méd. **38**:1552 (Nov. 15) 1930.

The clean, complete enucleation secured by the Ballenger-Sluder method is considered by Canuyt to be vastly superior to any other. He uses general or local anesthesia. The operation is rapid, but difficult for those unfamiliar with it. There is little bleeding, and the fossa is left clean, as the separation follows the plane of cleavage of the capsule from the fossa. This operation is greatly to be preferred in children.

BIEKER, St. Petersburg, Fla.

DIPHTHERIA: THE CAUSES AND PREVENTION OF THE MORTALITY IN DIPHTHERIA. F. SZIRMAL, Jahrb. f. Kinderh. **129**:293 (Nov.) 1930.

This is a careful report on 1,122 cases studied at the clinic of Professor Bókay from 1920 to 1928. The mortality in general was 12.3 per cent; excluding mixed infections, it was 9.7 per cent, and excluding 15 patients admitted in a moribund condition, 8.4 per cent. The mortality was 31 per cent in children under 1 year of age; 20.6 per cent, from 1 to 2 years, and 4.4 per cent from 7 to 14 years. Up to 1926, fatalities occurred chiefly in neglected nasopharyngeal diphtheria, and since 1926, in malignant diphtheria. In the earlier period, patients with mild cases received not more than 6,000 units of antitoxin, and those with severe cases, including croup, from 8,000 to 10,000 units; this dose was sometimes repeated. Since 1926, patients with mild cases received from 8,000 to 12,000 units; those with moderately severe cases, from 15,000 to 20,000 units, and those with severe or malignant cases, from 40,000 to 80,000 units. If the membrane spread, the dose was repeated. Antitoxin was given intravenously and intramuscularly. Between 1920 and 1925, paralyses occurred in 6.3 per cent of cases. In 44 per cent of the cases of croup, operation was avoided. The average mortality in the cases of croup was 22.8 per cent; this rate varied from year to year, from 6.6 to 29.5 per cent. The author strongly combats the opinion of Schlossmann that narcotics may be used instead of intubation. He feels, however, that they may be useful in avoiding reintubation. He is strongly in favor of primary intubation and has seen decubitus only once in nine years; this complication was frequent in Budapest in the nineties.

CLAUSEN, Rochester, N. Y.

ANGINA WITH A LYMPHATIC REACTION IN INFANCY. G. LEWIN, Monatschr. f. Kinderh. **48**:300, 1930.

In spite of the frequency of pharyngitis and otitis in infancy, tonsillitis is relatively rare. Lewin reports the case of an infant, aged 5 months, who was taken acutely ill with angina and membranous tonsillitis. Cultures showed hemolytic streptococci. The white count of 18,000 was almost entirely lymphocytes. Recovery took place.

While the author discusses the possibility of Pfeiffer's glandular fever, she prefers the diagnosis of angina with a lymphatic reaction.

GERSTLEY, Chicago.

ANGINA WITH ATYPICAL LYMPHOCYTEMIA IN CHILDHOOD. HERMANN SIEVERS, Ztschr. f. Kinderh. **49**:429, 1930.

A symptom-complex was described in 1907 by Tück under the name of "lymphatic reaction"; to this Sievers prefers "angina with atypical lymphocytemia." The children become sick either suddenly with pain in the head and high fever, or more insidiously with discomfort, dulness and slight fever. The sore throat varies in type; it may be catarrhal or diphtheroid, or in some cases necrotic. Moreover, there develops, usually early, a swelling of all the lymph glands, not merely the local ones, more or less enlargement of the spleen and in some cases enlargement of the liver. The red cells show no peculiarities. There is a leukocytosis of varied degree, from 9,000 to 3,000. The lymphocyte percentage may be from 50 to 90; the lymphocytes may show atypical forms; the monocytes may be increased and the polymorphonuclear cells correspondingly diminished. The blood platelets are normal in number or increased. The Wassermann reaction is negative and the blood culture usually negative. A special etiologic agent is unknown. The complications are otitis media, aphthous stomatitis and a rash. The duration is short, but the changes in the blood persist for months after the child is well. Sievers reports one case. The fact that three cousins died of acute lymphatic leukemia signifies a close relation between lymphatic leukemia and lymphatic reaction.

EMERSON, Boston.

Larynx

ACUTE LARYNGEAL OBSTRUCTION. LYMAN G. RICHARDS, J. A. M. A. **95**:766 (Sept. 13) 1930.

The author has evolved a plan of treatment in acute laryngeal obstruction which comprises careful consideration of the patient's condition and a decision as to whether or not there is improvement. If this does not occur in a reasonable length of time, laryngoscopy is indicated to determine the exact cause of the obstruction. Foreign bodies must be removed and if postoperative edema occurs, intubation may be necessary. In acute streptococcus laryngitis, intubation or tracheotomy is often necessary. The latter is more often used in order that further downward extension may be obviated. In diphtheritic laryngitis the membrane may sometimes be removed by suction. Intubation is the method of choice if this is possible, and the usual treatment for diphtheria should be instituted. In laryngitis sicca, suction will often remove the dry, grayish crust; if this is unsuccessful, intubation must be done. In obstruction due to extralaryngeal pressure, tracheotomy affords the best results.

BONAR, Salt Lake City.

NON-DIPHTHERITIC INFECTIOUS LARYNGITIS. G. M. CULTRA and A. J. STREIT, Texas State J. Med. **26**:364 (Sept.) 1930.

Thirteen definite, well formulated cases concisely given are the basis for this paper. The symptoms as listed are sudden onset, dyspnea and cyanosis, the condition being characterized by an exudate of thick, sticky brown mucus and laryngeal swelling. The cyanosis and dyspnea are caused by the bronchospasm and gluing together of the lumen.

In this series of cases the predominating organism obtained through the use of the laryngoscope and carefully cultured was *Staphylococcus hemolyticus.*

In severe cases, treatment consists of low tracheotomy and suction or mechanical removal of mucus. In milder cases, the administration of 1 drachm of sodium bicarbonate by mouth and the application of epinephrine or ephedrine to the congested mucous membranes through the laryngoscope give good results.

Antispasmodics, such as syrup of ipecac, are also valuable. Diphtheria antitoxin is used only in cases in which there is any doubt as to the bacteriologic diagnosis.

The authors vouchsafe the statement that this type of laryngitis is much more prevalent in the west and southwest than was formerly thought.

MOORE, Dallas, Texas.

Nose

ANALYSIS OF 100 CONSECUTIVE NASAL SINUS CASES TREATED CONSERVATIVELY. EUGENE R. LEWIS, Laryngoscope **40**:822 (Nov.) 1930.

The patients included in this series presented the following cardinal symptoms of sinus disease: (1) recurrent attacks of nasal inflammatory symptoms, (2) local pain, (3) mucoid or purulent nasal discharge, (4) hypertrophic, atrophic, fibrotic, polypoid or necrotic lesions of the mucosa, (5) turbinal turgescence, (6) gross architectural alterations of the nasal structures, (7) headache, frequently of the morning type, (8) aprosexia, (9) disturbance of the lower respiratory, the gastrointestinal, the glandular or the nervous system and (10) general debility. The majority of the patients had acute exacerbations of preexistent sinus trouble. Sixty-three of the patients had previously been operated on, from several months to several years before. Sixty-five were in possession of the tonsils. In thirty-nine cases, there was evidence of a special sensitivity of the tissue to foreign protein. On examination of eighteen patients, no pus was found in the nose. The ages varied from 7 to 72, the average being 37 years.

Under conservative treatment are included certain minor surgical procedures, such as: resection or reconstruction of the septum, simple incision of the antrum, bulla and sphenoid in order to give vent to purulent content, removal of polypi, tonsillectomy and adenoidectomy. The nonsurgical measures used included: suitable regular exercise, dietary regimen, periods of alkalization, periods of iodization, the use of salicylates, physical therapy, local therapy by tamponade, suction, oily sprays and the inhalation of steam, mechanotherapy, such as avoidance of blowing the nose vigorously, the avoidance of nasal washes or insufflation of water while swimming or bathing, and the continuance of such a regimen over long periods regardless of the presence or absence of symptoms.

After analyzing his results, the author found that they are definitely superior to those following radical surgical procedures in similar cases, the serious postoperative complications, such as osteomyelitis, meningitis, abscess of the brain, thrombosis and septicemia rarely being encountered in the use of the conservative method of treatment.

SNAPP, Grand Rapids, Mich.

VASOMOTOR RHINITIS. JOEL J. PRESSMAN, Laryngoscope **40**:878 (Dec.) 1930.

The diagnosis and treatment of vasomotor rhinitis has been unsatisfactory because of the inability to classify correctly the pathologic changes and to correlate these changes with the clinical observations. The author believes that one is much less likely to encounter error if one follows a definite routine examination based on known factors which tend to point out the presence of an underlying sensitization to protein. There are five ways by which an allergic turgescent rhinorrhea may be distinguished from a nonallergic form, namely: history, local examination, microscopic study of the nasal secretion, histologic examination of the tissue and allergic studies. A complete history is important and may lead, in itself, to a definite diagnosis. A careful study of the nasal passages is extremely necessary and may reveal much information. Of utmost importance is the microscopic examination of the nasal secretions for the presence of eosinophilic cells. The presence of a few eosinophils is suggestive of allergy, while the occurrence of large numbers is almost pathognomonic. The author believes that it is justifiable, in obscure cases, to remove a small piece of turbinate or mucous membrane for microscopic study. If the examination thus far reveals the condition as being due to allergy, the patient must be referred to an allergist for a differential diagnosis to determine the offending protein or proteins and for proper immunization. Fre-

quently, there is a combination of allergy and nasal pathologic changes which requires both allergic study and otolaryngologic treatment.

SNAPP, Grand Rapids, Mich.

MECHANISM OF PERCEPTION OF ODORS BY HEMATOGENIC ROUTE. M. BEDNÁR and O. LANGFELDER, Monatschr. f. Ohrenh. **64**:1133 (Oct.) 1930.

Perception of odors by the hematogenic route means irritation of the olfactory ends by odorous substances injected intravenously. The tests were made with (1) neoarsphenamine, with (2) a camphor preparation which has an intense odor of camphor, and (3) with a preparation containing 33 per cent of turpentine oil. In order to exclude the nasal perception of odor, tampons of gauze moistened with paraffin were inserted in the nose; they were removed as soon as the tested solution had been injected. Fourteen patients with an intact olfactory functioning have perceived the odor of all three of the test substances. Paradoxically, the odor was not perceived by a patient with an excessively developed sense of smell. The tests were negative in four patients with ozena associated with a pronounced atrophy of the nasal mucosa and with profuse crusts. In a syphilitic patient, who developed paralysis of the olfactory nerve after influenza, perception of odors failed by the nasal and by the hematogenic routes. The perception became restored for both routes as soon as the paralysis disappeared. In two patients with ethmoidal polypi, and in one with acute rhinitis, perception of odors was nil by the nose, while it was undisturbed by the hematogenic route. The latent period, extending from time of the injection to that of perception of the odor, varied from six to seven seconds. This is the time within which an odorous substance injected into the median ulnar vein reached the olfactory region. The mechanism of perception of odors by the way of the circulating blood is explained by a direct action of odorous molecules, resulting in an intramucosal irritation of the olfactory endings. The neurogenous nature of the hematogenic perception of odors is proved by the fact that it was not impaired when a complete obstruction of the nose by polypi was present. The odor of the injected substances was clearly perceived when simultaneously menthol or paraffin were instilled in the nose, or when benzine was smelled. Spraying of the olfactory area with cocaine and epinephrine resulted in impairment of smelling by the nose, while the perception of odors by the hematogenic route was not affected. It appears that there was rather failure of combination of the odorous molecules with cellular lipoids which usually results in irritation of the olfactory center, than paralysis of the olfactory endings. Obviously the odorous substances could not have reached the olfactory mucosa and be eliminated there, because the latent period was too short. The fact that nasal tampons withdrawn after the injections were odorless contradicts the theory that the nasal mucosa possesses an excretory functioning. Moreover, no traces of arsenic were found in tampons introduced in the nose during injection of neoarsphenamine. Treatment of the olfactory mucosa with atropine failed to inhibit the sense of smell or to prolong the latent period. After repeated injections of an odorous substance, the perception of the odor decreased, while the perception of odor appeared normal when another substance was then injected. Thus the fatigue of the olfactory nerve proved to be specific. Two patients have had the sensation of tasting neoarsphenamine after the injection; no sensation of taste occurred after injection of the two other substances.

EDITOR'S ABSTRACT.

Miscellaneous

RADIUM TREATMENT OF CANCER OF THE TONGUE. STANFORD CADE.

RADIUM AND SURGERY IN CANCER OF THE TONGUE. DOUGLAS QUICK.

THE PRINCIPLES OF RADIUM TREATMENT OF BUCCAL CARCINOMA. G. E. BIRKETT, Canad. M. A. J. **23**:771, 774, 780 (Dec.) 1930.

The three writers agreed that radium was the method of choice in treating carcinoma in the mouth. For the primary growth, Cade and Birkett preferred

radium needles, and Quick favored radon seeds. The first two men claimed it was difficult to obtain a uniform intensity of radiation with seeds, not only because the intensity of the radiation rapidly diminished within a period of four days, but also because the small seeds could not be so accurately placed as the larger needles. For the treatment of metastases to the neck that were operable, they all favored surgical excision, and stressed the importance of radical removal, for an incomplete dissection was worse than useless. In the case of large inoperable metastases, the glands should be actively treated by radium needles in large doses, and the treatment should be carried out by open operation. For early cases with no palpable metastases, there was difference of opinion. Birkett advocated a waiting policy with frequent observations of the patient. Cade and Quick believed that the neck should never go untreated. Cade advocated treatment of the neck with needles by the closed method or external treatment by means of a radium collar. Quick employed heavily filtered high voltage x-rays. As a preliminary to irradiation of the primary tumor, he also recommended external irradiation of the neck, including the site of the primary growth for two weeks. Their results clearly indicated the superiority of radium treatment over treatment by surgical intervention alone, for there was no mutilation and no impairment of function.

RATTNER, Chicago.

STUDIES OF THE ETIOLOGY OF THE COMMON COLD. GERALD S. SHIBLEY, KATHERINE C. MILLS and A. R. DOCHEZ, J. A. M. A. **95**:1553 (Nov. 22) 1930.

The authors conclude from their studies that a contagious cold is caused by an invisible, uncultivable, filtrable agent which probably belongs to the submicroscopic viruses. It can be transmitted by means of Berkefeld filtrates of nasal washings obtained from persons suffering from colds, and the colds so contracted appear clinically the same as those contracted under natural conditions. In apes the infection stimulates any potential pathogens present to greatly increased activity, a fact that explains the marked secondary activity of the pneumococcus, *Streptococcus hemolyticus* and Pfeiffer's bacillus, which often leads to severe sequelae in common colds. The most important significance of the virus seems to be this catalyzing action.

BONAR, Salt Lake City.

BRASS PAPER FASTENER IN BRONCHUS. M. P. STILES, South. M. J. **23**:1113 (Dec.) 1930.

A brass paper fastener was found in the bronchus of a boy, aged 11 years. He had the fastener in his mouth and accidentally aspirated it. In spite of the fact that it was double-pronged, its removal was accomplished without unusual difficulties.

SCHLUTZ, Chicago.

THE INDICATIONS, TECHNIC AND ACCIDENTS OF TRACHEOTOMY. O. ROSSERT and A. MOATTI, Paris méd. **2**:200 (Sept. 6) 1930.

The indications for tracheotomy in the child are: laryngeal dyspnea from croup or diphtheria, in which intubation has become unsatisfactory through repeated retubings, laryngitis from contagious diseases and papilloma of the larynx. Indications for tracheotomy in adults are also given. The method advised is that of tracheal fixation. The immediate accidents are: arrested respiration, usually induced in the last stages of a severe disease, and hemorrhage from wounding the great vessels. The complications are infection and bronchopneumonia.

SANFORD, Chicago.

Society Transactions

GERMAN OTOLARYNGOLOGIC SOCIETY

Basle, Switzerland, June 5, 6 and 7, 1930

ABSTRACTED BY ALFRED LEWY, M.D., CHICAGO

(Ztschr. f. Hals-, Nasen- u. Ohrenh. **27**:329-642, 1930)

PHYSIOLOGY OF HEARING: EXPERIMENTAL INVESTIGATION OF THE MECHANICS OF THE OSSICULAR CHAIN, AND ITS BEHAVIOR UNDER TONE AND AIR PRESSURE IMPULSES. DR. H. DAHMANN.

The author completes his investigations and report on the results obtained and conclusions made. By means of tiny mirrors fastened on the ossicles and tympanic membrane, which reflected a beam of light to sensitized paper on a kymograph, he demonstrated their movements under various conditions, all of which was reported at the congress last year (abstr., ARCH. OTOLARYNG. **11**:239 [Feb.] 1930). This year he divides his article into three parts, dealing with: (1) the accuracy and mathematical proof of his observations; (2) the statics and dynamics of the conduction apparatus; (3) a theoretical and practical discussion of the hearing phenomena. The fundamental idea is that a sound wave is primarily received by bone conduction and meets another sound entering via the conduction apparatus with a different phase, therefore acting as a damper, and to the degree that this damping effect is lost through defect of the conduction apparatus bone conduction is lengthened. (No mention is made of the experiments by Kranz and by Knudsen in this country which show no prolongation of bone conduction in a sound proof room, and attribute the apparent prolonged bone conduction to the absence of masking by outside sounds.)

MECHANICS OF BONE CONDUCTION AS SHOWN BY A MODEL. DR. HERZOG.

By direct impact of sound waves there ensues a rhythmic movement of the fluid toward both windows, which is greater toward the round window. It is through this impact that the conditions for the transverse vibrations of the membrana basilaris are brought about.

FUNCTIONAL EXAMINATION OF THE OPENING AND CLOSING OF THE TUBA AUDITIVA, AND SOME PHYSIOLOGIC AND PATHOLOGIC OBSERVATIONS. DR. VON GYERGAY.

The author describes both old and original methods for the examination of the movements and functional ability of the tube. One of the methods devised by him is the introduction of a tiny lamp into the tube, the light of which may be observed through the membrana tympani during movements of the tube induced by swallowing. The author hopes to unravel some of the mysteries of the complicated movements of the eustachian tube.

FURTHER OBSERVATIONS ON THE RELATION BETWEEN ENDOLYMPHATIC PRESSURE AND THE HEARING FUNCTION. DR. KEREKES.

The author used injections of epinephrine to increase the blood pressure. In all cases in which there was an increase of over 30 mm. there was diminution of

bone conduction, and in one case definite loss of hearing for speech. In five cases in which trepanation of the skull was done, pressure on the exposed area caused decreased hearing in four and in one improved it for the whisper, with improvement of previously shortened bone conduction.

PHYSIOLOGY OF THE INTRINSIC MUSCLES OF THE EAR: INVESTIGATIONS OF THE MECHANICS OF THE OSSICULAR CHAIN; DEMONSTRATION. DR. KOBRAK.

It can be demonstrated in animals that a tone produces in the intrinsic muscles tetanic contractions which can be measured. The strength of the contractions is dependent on intensity and frequence. The strongest contractions are produced by tones that resonate with the fundamental tone of the ossicular chain. This was tried on fresh human specimens. Weak stimuli caused throttling at the stapes alone by the stapedius, while loud tones caused damping, first at the malleus and again at the stapes. Investigation of the fundamental tone of the conduction apparatus, in spite of variation of its susceptibility by the muscles, showed that it maintained its resonance curve. Searching investigations showed that the ossicular axes as well as the leverage transposition of the conduction apparatus are changed by the muscle pull. This indicates that investigations of the pathologic physiology of the intrinsic muscles is of clinical value.

CONTRIBUTION TO THE PHYSIOLOGY OF HEARING. DR. ALEXANDER.

The author analyzes the known facts and theories in regard to air conduction, presenting those for and against conduction via the ossicles and via the air chamber of the tympanic cavity and the membrane of the round window. He divides his statements into clinical observations, pathologico-anatomic facts, embryologic indications and comparative anatomy. He appears to favor the idea of tympanic cochlear conduction via the membrana secundaria. One argument against tympanocochlear conduction which he fails to note is the difficulty of transmutation of sound waves from air to water medium.

THE FUNCTION OF THE INTRINSIC MUSCLES OF THE EAR. DR. FREY.

The present views of the function of the intrinsic muscles of the ear are unsatisfactory as they do not appear to meet the anatomic situation, nor do they fulfil physiologic requirements. The idea that they form an accommodation mechanism is weakened by the fact that such an occurrence has never been observed during the act of hearing. Observations on animals, on cadavers and on living persons have been made under conditions that bring about overactivity. The author believes that anatomic conditions and experimental facts show that the normal function of the intrinsic muscles is to provide elasticity, as well as to preserve the plane of movement in the ossicular chain, through their constant tonus, and that only in case of overirritability or unusual stimulus do the muscles act as a defense mechanism. It is difficult to compare hearing in animals with that in man, as one is hardly acquainted with the act of hearing in animals, and it may have entirely different connotations than in man.

DIFFERENCES IN PITCH BETWEEN TINE AND STEM TONES IN WEIGHTED "OVERTONE-FREE" TUNING FORKS, A SOURCE OF ERROR IN THE RINNE TEST. DR. FRENZEL.

A number of commercial weighted *C* forks (64 double vibrations) were heard by bone conduction in the first overtone, viz., *c* (128 double vibrations), the same fork being heard by air in its fundamental tone *C*. As a cause, Frenzel assigns the difference in length of vibration between the tines and the stem. As this fork is used by some for the Rinne test, it may lead to error, for in inner ear deafness with loss of low tones the error may lead to the perception of Rinne negative; *C* heard by air conduction may be compared with *c* heard by bone conduction.

HEARING FOR A WHISPER AND FOR A CONVERSATIONAL TONE (A CONTRIBUTION TO THE QUESTION OF TONE HEARING, SPEECH UNDERSTANDING AND FORENSICS). DR. NADOLECZNY.

It is well known that in inner ear deafness a conversational tone is heard much better than a whisper, while in typical otosclerosis there may be little if any difference. This is because in inner ear deafness tones are poorly heard in which lie the formants of the higher vowels and consonants. There are also cases in which tests with numbers give good results in spite of considerable loss of hearing for the low tones, because the formants for these lie within the good area while the region in which lie the pitch of the whisper vowels has suffered. In a still higher region the sibilants may again be heard better. The point is that the ordinary conversational tone is the practical test for forensic purposes, and that scientific tests of the hearing acuity are not practical for this purpose.

The author looks on these experiments as supporting the investigations of V. Bekesy, who found displacement of the footplate of the stapes either inward or outward, according to the minus or plus pressure of endolymph. This experimental displacement of the stapes always causes diminution of hearing in normal persons. The author concludes that there is an optimum endolymphatic pressure when hearing is good, and that deviation in either direction from this optimum decreases the hearing.

THE INFLUENCE OF OUTSIDE NOISES ON THE PERCEPTION OF SOUND. DR. HIRSCH.

The hearing of normal persons and of others with decreased hearing was tested by speech, tuning forks and electrically produced tones. For masking purposes, other noises (machinery, music and whistle) were used. It was found that these noises masked the hearing in varying degrees for whisper, tuning forks and electrical tones, and that the masking effect was much greater in patients with inner ear deafness than in those with conduction deafness.

THE PRINCIPAL BASES OF A NEW AID TO HEARING BY THE USE OF THE BONE CONDUCTION. DR. SONNTAG.

The instrument consists of a microphone, conductor and a bone telephone, and is placed on the skull behind the ear. The vibrations are transformed without much amplification. The device is demonstrated to improve the hearing in conduction deafness.

THE INFLUENCE OF VERY SHORT ACOUSTIC WAVES ON THE INNER EAR. DR. MUELWERT.

The author experimented with high frequencies (from 30 to 34,000 cycles) and obtained an increase in hearing for low tones. As to the danger of injury by these high frequencies above the limit of conscious hearing, he states that after four years of much application to himself there were no ill effects.

THE FUNDAMENTAL TONE OF THE CONDUCTION APPARATUS AND THE RINNE TEST. DR. KRAINZ.

According to O. Frank, the fundamental tone lies at about 1,200 cycles. In tension of the chain it is higher; by increasing its mass, it is lower. If the chain is free, bone conduction is shorter, being damped by the phase difference between the chain and labyrinthine vibrations. The nearer the tone is to the fundamental tone of the chain, the greater the amplitude and therefore the greater the relative duration of air conduction. If the frequency of the chain is decreased but its mobility not interfered with, as in the water-filling experiment, the Rinne test for low tones in the neighborhood of the maximum resonance may be negative, but for the higher tones positive.

Speech and Letter Understanding, and Its Relation to Hearing of Speech in Deaf-Mutes. Dr. Grahe.

Fifty-four deaf-mutes (108 ears) were examined; 40 (37 per cent) were deaf, and 68 (63 per cent) had some hearing. The results of the tests of hearing varied greatly. Eleven persons could hear only tone, mostly in the high registers; 4 could hear syllables; 32 could hear speech at the ear but could not understand it. Of these, 11 could hear no single syllables, but 2 could distinguish them. Twenty-one understood words and sentences at the ear. One of the 21 heard all vowels and selected consonants; others could hear some consonants and usually vowels. Altogether, the cases showed no agreement with the Bezold rule that the pitch between b^1 and g^2 must be heard at least 10 per cent in order to have understanding of speech. On the other hand, there was agreement with theory of formants as established by Stumpf and others in the sense that a certain syllable connoted hearing for a particular range, but the reverse was not true that with hearing for certain pitches speech could be heard.

Contribution to the Pathologic and Clinical Signs in the Central Hearing Tracts. Dr. Brunner.

Brunner demonstrated specimens of the organ of hearing in a case of acquired deafness and in a case of subcortical sensory aphasia, and discussed the condition of the brain in these cases.

Injuries of the Ear at Birth. Dr. Albrecht.

The author examined the temporal bones of 80 stillborn children (160 in all). He corroborates Voss' observation that bleeding into the ear is common. As the vestibule is seldom affected, and only the perilymphatic space, serious injury is avoided. Cochlear bleeding was also confined to the perilymphatic spaces, except in one case in which it occurred in the ductus cochlearis. At the entrance point of the nerve and vessels at the modiolus in the porus acusticus, hemorrhage was frequent, the nerve was often seriously damaged, and even the spiral ganglion was involved. In the middle ear, bleeding of various degrees was the rule. In the cases in which the embryonal tissue had been absorbed, no particular damage was done, but in the cases in which the ears were still filled with embryonal tissue, which was common, there was considerable disturbance of this tissue, with inflammatory reaction in the contiguous areas, laying the groundwork for the so-called suckling otitis. Bleeding was more frequent and more severe in premature infants, so that the development and permeability of the vessels appear to be factors. The tendency for bleeding to occur at the entrance point of vessels into bone appears to indicate this as a locus minoris resistentiae. Albrecht concludes that injury at birth may cause serious bleeding, and while this should not be overemphasized, neither should it be underestimated.

Displacement and Kinking of the Esophagus. Dr. Beck.

A woman, aged 40, with history of pleurisy eight years before, had for sometime had dysphagia for solids but not for liquids. An esophagoscope was stopped at the level of the bifurcation, but showed no change in the mucosa; there was only a blank wall. By introducing a bougie, the esophagoscope could be passed to the cardia. A roentgenogram showed a corkscrew-like winding at the level of the bifurcation. The esophagus and trachea in their upper part were drawn to the right. The right half of the thorax was shrunken and much smaller than the left. The ribs ran vertically and close together; the diaphragm was high and adherent. There was no evidence of tumor. This is a case of extensive contraction with adhesions and deformity following pleuritis.

DIFFERENTIAL DIAGNOSIS BETWEEN FOREIGN BODY AND CARCINOMA OF THE ESOPHAGUS. DR. HIRSCH.

A clinical diagnosis of carcinoma was made in a man, aged 68, with extreme cachexia; esophagoscopy was not risked. After roentgen examination gastrostomy was done. In fourteen days the patient was much better, and esophagoscopy was undertaken, which revealed a large piece of bone which the patient could not remember swallowing. Removal was easily accomplished. The roentgenogram showed that the test meal had flowed on either side of the obstruction, which would manifestly be impossible in case of a tumor.

CLOSURE AT THE MOUTH OF THE ESOPHAGUS AFTER THORACOPLASTY FOR STENOSIS. DR. KELEMEN.

The author describes the plastic operation of Prof. V. Bakay (Budapest), which is done in five stages: (1) the stump of the esophagus is brought out in the left side of the neck above the clavicle; (2) a tube is constructed from the skin of the breast; (3) a loop of jejunum is brought out and jejunostomy is performed; (4) the neck end of the esophagus is joined with the skin tube; (5) the skin tube is joined with the opening in the abdominal wall. Sixteen such operations have been completed, and many others are in various stages of completion. Valuable observations have been made, especially in regard to closure of the esophagus against regurgitation; it can be closed so tight that if vomiting occurs it constitutes a great drawback, being unregulated from the cardia.

ENDOSCOPIC TREATMENT OF CARCINOMA OF THE ESOPHAGUS WITH DIATHERMY. DR. HESSE.

Too high a mortality results from surgical treatment, and irradiation is only temporarily successful; therefore, the author used electrocoagulation in six cases. Severe stenosis was present in the first case and bougies had to be used before treatment could be given. Short rod-shaped or spherical electrodes are best, the thickness varying with the degree of stenosis. The electrodes are fastened to well insulated handles of proper length to reach the lesion. Intensive coagulation was well borne by the patients, and the danger of periesophagitis seems to be slight. So far this has not been observed, nor has there been cicatricial contraction.

SPONTANEOUS CURE IN PERFORATION OF THE ESOPHAGUS AND MEDIASTINAL ABSCESS. DR. HERRMANN.

The author has revised his idea that the prognosis in these conditions is always grave, especially as Seifert reported several cases with recovery. In the last year two such cases occurred in his clinic. In one case, that of a boy, a perforation in the lower part of the esophagus occurred following bougienage for stenosis, due to the ingestion of lye, and a mediastinal abscess resulted. In the other case the mediastinal abscess resulted from perichondritis of the trachea, with spontaneous rupture into the esophagus. Both patients recovered. Roentgenograms are shown.

PROPHYLAXIS OF ESOPHAGEAL STRICTURE FOLLOWING CAUSTIC ACCIDENTS. DR. HOFFMANN.

In a survey of ten years' experience with the Salzer method of early bougienage, 153 cases are reported, and comparison is made with other methods of treatment. Most of the patients were 2 or 3 years of age. Recovery occurred in 89 per cent. Eleven per cent were fatal, in 2 per cent of which death was due to the bougienage. The author believes this indicates early bougienage, at least so far as children are concerned.

TREATMENT FOR CORROSIVE ACUTE AND CHRONIC ESOPHAGITIS. DR. BELINOFF.

Belinoff is in favor of roentgen examination and esophagoscopy to determine treatment and the right time to begin bougienage. He distinguishes four stages: necrosis, ulceration, granulation and cicatrix. He has devised a syringe on the cannula of which is a balloon tampon, which is filled with air, and which protects the walls against trauma while the injection is being made. Oily solutions are used until healing reaches the proper stage for bougienage if this is necessary. In chronic cases dilation is done with metal dilators.

IS THE GOLDMANN OPERATION FOR DIVERTICULUM OF THE HYPOPHARYNX TO BE RECOMMENDED? DR. OPPIKOFER.

The author obtained good results with this method in the first six cases. In the seventh case, there was rapid necrosis, which extended to the carotid wall, with thrombus and embolus to the sylvian artery. Death occurred on the tenth day.

RELATIONS OF THE DISEASES OF THE TEAR SAC TO THE NOSE, AND THEIR TREATMENT. DRS. KOFLER and MEESMANN.

Accurate orientation of the anatomic construction and topographic relations of the tear ducts, as well as knowledge of the frequent anomalies of this region, is necessary for critical judgment and indications for the various operative and conservative measures. The most frequent causes of diseases of the tear ducts are found in the nose, less often in the eye and least of all from conditions of other surrounding structures (traumas, diseases of the bone). The principal cause of dacryocystitis is ascending infection combined with mechanical obstruction. For this reason the best prophylaxis is proper treatment of the nose. The frequent relationship of diseases of the tear ducts and of the nose presents the desirability of intimate cooperation between ophthalmologists and rhinologists, not only for the determination of a nasal cause, but for the election of endonasal or paranasal approach. The author describes indications for the various procedures (extirpation of the sac from without, internal and external dacryocystorhinostomy, etc.) their advantages and disadvantages, possible complications and results. Comparing the operations of West and Toti, the author concludes that it is better to use the internal route than the external one on account of the following disadvantages: dangers from the skin incision (scar, keloid); possibility of infection of the wound (erysipelas); operative difficulties on account of variations in the fossa; impossibility of the modified Toti operation in atrophy of the sac or in tuberculosis; injury to the surrounding musculature; difficulty and danger of external operation in dacryophlegmon; greater difficulty of exenterating adjacent ethmoid cells, which is more easily done via the nose. On the basis of his 160 endonasal operations, the author advises: accurate knowledge of the topography of the region of the tear sac and its variations; a good view of and a wide approach to the operative field, which is best achieved by the transseptal route; good anesthesia; incision so that the base of the flap lies on the upper border of the concha inferior; removal of the bony fossa to the top of the tear sac; exenteration of all overlying or bordering ethmoid cells; complete removal of the medial wall of the tear sac; seldom resection of a too large anterior end of the middle concha; tamponade of the wound of the tear sac for at least eight days; prolonged control after the operation. Statistics of the results of various authors with both operations are given, also a bibliography.

RESULTS OF ENDONASAL OPERATIONS ON THE TEAR SAC (EXAMINED FUNCTIONALLY BY X-RAYS). DR. UNTERBERGER.

The author shows results in thirty-seven cases in which operation was done by the endonasal route, and concludes that the statistics given by Kofler are too favorable. Functional tests were made by injections of iodized sesame oil and roentgenograms made immediately after the injections and also in fifteen or

twenty minutes. In normal persons the pathways are practically clear in from two to five minutes. In cases of relative and complete stenosis the tear sacs are completely or partly filled after fifteen or twenty minutes, and in some it takes fifteen or twenty minutes to fill the sacs in the first place. The author reports ideal results in 25 per cent; functionally good but some stenosis discernible in 50 per cent; partial success (overflow of tears, but no pus) in 75 per cent; failures in 25 per cent. Contraindications are diseases of the eye itself, especially those in which sterile operation on the eye may become necessary.

WHAT DOES MY INTRANASAL OPERATION ON THE TEAR SAC ACCOMPLISH? DR. HALLE.

The author gives a historical review of the development of this operation beginning with the original one of West, and compares it with the external operation as performed by ophthalmologists. He states that the intranasal operation is practical from infancy to old age, and is in the highest degree physiologic; it is to be compared only with Toti's operation; it has the advantage over this operation in that it can be performed in all cases regardless of fistulas, phlegmon, infections of the skin, and other conditions without leaving an external scar. In properly performed operations, properly controlled, one can expect 100 per cent of healing in a week.

SEVEN YEARS' EXPERIENCE WITH THE GLASS PROSTHESIS OF THE LOWER TEAR CANAL. DR. HEERMANN.

A well performed operation with West or Toti's methods, which fails to restore proper tear drainage, is an indication for this prosthesis. The author describes his cannula and its application. It can be used as long as the anatomic relations of the inner canthus are not destroyed, therefore, after extirpation of the sac, phlegmons, etc. If the lower canal is suppurating or has been opened too widely, there may be difficulty in retaining the cannula. It is also difficult if there is no stump of the canal left so that the cannula can be surrounded by mucosa, but must lie free in other tissues. All other cases permit of free drainage of tears and healing of suppuration.

A HITHERTO UNOBSERVED ANOMALY OF THE MEMBRANOUS LABYRINTH. DR. FISCHER.

Fischer describes a specimen which showed a third macula acustica and gives a complete description of the associated anatomy, some of which resemble that in lower animals. He concludes that from a physiologic standpoint these anatomic features in man represent either a higher development of function or an atavism.

EXPERIMENTAL INVESTIGATIONS WITH QUININE. DR. SEIFERTH.

The effect of quinine on the ear was tried on dogs. No definite pathologic changes could be demonstrated with certainty. A few dogs showed changes in the ganglion cells and the sheaths, consisting of indistinct contours and poor staining, and pyknosis of the nuclei of some of the cells. These changes cannot with certainty be described as pathologic. In view of the fact that quinine belongs to those substances to which some people have an idiosyncrasy, the author asks whether the disturbances in hearing following ingestion of quinine are always toxic or whether they may not be due to idiosyncrasy.

EXTRAORDINARY BEHAVIOR OF THE PULSE IN DISEASES OF THE LABYRINTH AND AFTER RADICAL OPERATION. DR. BECK.

The author publishes graphs of the pulse rate in several cases in which it varied from very slow to very rapid, without relation to the temperature. Brady-

cardia has been described by Neumann and Borries in connection with labyrinthitis. Spiegel denies reflex action and attributes the slowness to associated pressure of the posterior fossa. Beck ascribes the bradycardia to sympathetic irritation in the tympanic plexus, which may either slow the pulse or quicken the heart, according to which impulse outweighs the other.

CONTRIBUTION TO THE CLINICAL AND PATHOLOGIC SIGNS IN ABSCESS OF THE BRAIN. DR. NEUMANN.

Neumann raises the question if in recent years the advances in diagnosis, that is, of new symptoms, has led to any improvement in operative results, or whether waiting for more conclusive evidence has led to fatal delay. Formerly, when general symptoms were sufficient to warrant operative exploration, statistics would indicate better results. Neumann notes earlier intervention in cases of right-sided temporosphenoidal abscess than in left-sided, since the operation on this side is often delayed for the appearance of the characteristic aphasia, a late symptom. He discusses the difficulty of evaluation of statistics because of lack of essential details. He dwells on the importance of experience in handling these cases, especially in the after-care, and seems to favor the filiform bougie method of Lemaitre as the primary drainage method, to be modified according to developments in each case. Whether the greatest emphasis should be placed on drainage and after-care or on the time of operation is almost impossible to say. The best operative methods are those which achieve healing without leaving the patient permanently disabled. He especially mentions patients with healed abscess who, by reason of a large prolapse, are socially and psychically crippled.

THE VALUE OF THE BULBUS OPERATION. DR. SCHLANDER.

The author analyzes the statistics of various clinics in evaluating the results of the bulbus operation, i. e., the complete conversion of the lateral sinus into a gutter (Grunert) as against drainage of the lateral sinus, plus jugular section and careful irrigation after softening of the thrombus takes place, as practiced in the Neumann clinic. He finds the latter procedure fully equal to the complete opening, which is reserved for those cases in which pathologic bone extends to the region of the bulb and therefore necessitates exenteration.

NYSTAGMUS ON CHANGE OF RECUMBENT POSITION. DR. RUTTIN.

Ruttin describes three groups of cases in which this condition occurs:

The first group consists of cases in which nystagmus and vertigo develop when the patient is lying on one side and the head is rotated or inclined toward that side. These persons often show slight nystagmus when lying on the opposite side, getting up or lying down, inclining the head forward or backward. The nystagmus may be evoked by either fast or slow movement, but ceases or is of short duration (few seconds) when the patient comes to rest in any of these positions.

The second group consists of cases in which marked nystagmus and vertigo are evoked to the same or opposite side under the foregoing conditions, and which also ceases when the patient comes to the resting position, but only after several minutes. Usually this nystagmus is distinctly one-sided, i. e., it occurs only when the position or movements are on one side.

The third group is made of cases in which various positions of the head or body may evoke nystagmus and vertigo, without any definite rule. A number of these are described in detail.

A considerable bibliography is appended.

EXPERIMENTAL STUDIES REGARDING THE PATHOLOGIC PROCESS IN MENINGITIS. DR. POPPER.

By injections into rabbits, the author studies the diffusion of fluids in the subdural space, and the spread and distribution of bacteria in meningitis. He comes to the conclusion that in order to reach the bacteria it is necessary to medicate via the blood stream as well as subdurally.

THE QUESTION OF NARCOSIS FOR TONSILLECTOMY. DR. SPIES.

The author recommends the hanging head position, and uses an ether chloroform mixture which is administered through a flat tube tongue depressor attached to the head; a tube through the nose gives continuous suction, and a suction tube is used by hand in the cheeks and fossae as necessary. The anesthetist is not needed when a Braun narcosis apparatus is used. Spiess advises against "avertin" and "pernocton," because of the prolonged sleep and danger of aspiration. Various modifications of this technic appear in the discussion.

THE SCHOENBORN-ROSENTHAL OPERATION FOR CLEFT PALATE FROM THE STANDPOINT OF THE SPEECH SPECIALIST. DR. SCHILLING.

Schilling recommends the Schoenborn-Rosenthal operation from the functional standpoint. It consists of a pedicled flap from the posterior pharyngeal wall which is laid into the defect in the palate. In the discussion, the author contends that he has seen no ill effects from the partial closing off of the nasopharynx.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. T. E. Carmody, 227, 16th St., Denver.
Secretary: Dr. Gabriel Tucker, Bronchoscopic Clinic, University Hospital, Philadelphia.
Place: Philadelphia. Time: June 8-12, 1931.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
President-Elect: Dr. J. F. Barnhill, Miami Beach, Fla.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: French Lick, Indiana. Time: Sept. 14-19, 1931.

American Bronchoscopic Society

President: Dr. Henry B. Orton, 24 Commerce St., Newark, N. J.
Secretary-Treasurer: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.
Place: St. Louis. Time: June 6, 1931.

American Laryngological Association

President: Dr. Francis R. Packard, 304 S. 19th St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Place: Atlantic City, N. J. Time: June 15-17, 1931.

American Laryngological, Rhinological and Otological Society

President: Dr. Max A. Goldstein, 3858 Westminster Pl., St. Louis.
Secretary: Dr. R. L. Loughran, 33 E. 63d St., New York.

American Otological Society

President: Dr. David H. Walker, 101 Bay State Rd., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

SECTIONAL

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. G. G. Marshall, 122 West St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

New England Otological and Laryngological Society

President: Dr. Harry P. Cahill, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Chester H. Bowers, 1136 W. 6th St., Los Angeles.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

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* Secretaries of societies are requested to furnished the information necessary to make this list complete and to keep it up to date.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Albert E. Hillis, 1115 Pacific Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Fletcher D. Woodward, Box 162, University, Va.
Secretary: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: New Orleans. Time: November, 1931.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Wilfrid Haughey, 65 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. H. Fraser, Battle Creek Sanitarium, Battle Creek.
Time: Third Thursday of alternate months.

STATE

COLORADO OTO-LARYNGOLOGICAL SOCIETY

President: Dr. C. E. Cooper, 227, 16th St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Dorland Smith, 836 Myrtle Ave., Bridgeport.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. William L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Gordon F. Harkness, 215 Main St., Davenport.
Secretary-Treasurer: Dr. W. F. Boiler, 105 Iowa Ave., Iowa City.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John C. Brown, 350 St. Peter St., St. Paul.
Secretary-Treasurer: Dr. Hendrie W. Grant, Lowry Bldg., St. Paul.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Lee W. Smith, 125 W. Granite St., Butte.
Secretary: Dr. J. G. Parsons, Lewistown.
Time: Semi-annually.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. B. Beeson, 232 W. De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. W. McCollom, 346½ Washington St., Portland, Ore.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland, Ore.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Raymond F. Hacking, 105 Waterman St., Providence.
Secretary: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Fletcher D. Woodward, Box 162, University.
Secretary-Treasurer: Dr. Henry Grant Preston, Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. S. S. Hall, Fairmont.
Secretary: Dr. G. A. Smith, Montgomery.
Place: Clarksburg. Time: May 19-21, 1931.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. J. Wallace Hurff, 86 Washington St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. John R. Childs, 45 Edgewood Ave., Atlanta.
Secretary: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE CITY MEDICAL SOCIETY, SECTION ON OTOLOGY AND LARYNGOLOGY

Chairman: Dr. William R. McKenzie, 11 E. Chase St., Baltimore.
Secretary: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. George J. Dennis, 25 E. Washnigton St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

CINCINNATI OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Edward D. King, 707 Race St., Cincinnati.
President-Elect: Dr. M. F. McCarthy, Union Central Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:45 p. m., second Tuesday of each month from October to May.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. William V. Mullin, 9204 Euclid Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 2064 E. 9th St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

College of Physicians of Philadelphia, Section on Otolaryngology

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. A. M. Hauer, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. H. B. Decherd, 1717 Pacific Ave., Dallas, Texas.
Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Detroit Oto-Laryngological Society

President: Dr. Don A. Cohoe, 13535 Woodward Ave., Detroit.
Secretary-Treasurer: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. J. J. Richardson, 606 Penn St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robinson, Eye, Ear, Nose and Throat Hospital, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. E. L. Lingeman, 241 N. Pennsylvania St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. A. E. Eubank, Arygle Bldg., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Dean E. Godwin, Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Ferris Arnold, Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

President: Dr. Eugene R. Lewis, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Hospital, assembly room. Time: 8 p. m., first Monday of of each month, May to November, inclusive.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. John M. Auwerda, 878 President St., Brooklyn.
Secretary-Treasurer: Dr. Charles R. Weeth, 37 Remsen St., Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W.: Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

The Milwaukee Oto-Ophthalmic Society

President: Edward R. Ryan, 425 E. Water St., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 120 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville.
Secretary-Treasurer: H. C. Smith, Medical Arts Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. W. R. Buffington, 211 Camp St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Laryngology and Rhinology

Chairman: Dr. John M. Loré, 240 West End Ave., New York.
Secretary: Dr. Robert E. Buckley, 2 E. 103d St., New York.
Place: 103d St., and 5th Ave. Time: 8:30 p. m., fourth Wednesday of each month from October to May.

New York Academy of Medicine, Section of Otology

Chairman: Dr. Clarence H. Smith, 2 E. 54th St., New York.
Secretary: Dr. James Garfield Dwyer, 57 W. 57th St., New York.
Place: 103d St. and 5th Ave. Time: Second Friday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. Charles M. Swab, Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. J. C. Davis, 1615 Howard St., Omaha.
Place: Medical Arts Tea Room. Time: 5:15 and 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Arthur J. Wagers, 1429 Spruce St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. Watson Marshall, 121 University Pl., Pittsburgh.
Secretary-Treasurer: Dr. Thomas B. McCollough, 121 University Pl., Pittsburgh.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. Clyde Heatly, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 S. Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. George E. Hourn, 607 N. Grand Blvd., St. Louis.
Secretary: Dr. Arthur M. Alden, Frisco Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. T. J. Walthall, 728 Main St., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Wilber F. Swett, 693 Sutter St., San Francisco.
Secretary: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. Paul A. Remington, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Searle B. Marlow, State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubinstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

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